PUBLISHED WEEKLY

PRICE TWO SHILLINGS AND SIXPENCE

THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—42ND YEAR SYDNEY, SATURDAY, SEPTEMBER 10, 1955 No. 11

Registered at the G.P.O., Sydney, for transmission by post as a newspaper.

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Table of Contents.

IT COULD HAPPEN TO US.[1]

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I WILL begin this lecture with two quotations:

> Only the Devil could have devised this Godless weapon. Mankind can never withstand the blinding flash the seering ball. Its invention means the end of humane nobility.

> Prompted by widespread fears that this new weapon of mass destruction might wipe out Western civilisation the Pope today issued a Bull forbidding their use by any Christian state against another whatsoever the provocation.

It all began at 0815 hours on August 6, 1945. A few minutes earlier an enemy aircraft had been seen flying at a height of over 20,000 feet. As the aircraft neared the centre of the city, a small parachute was released bearing a burden. "Only more propaganda leaflets" was the general opinion, and the trams, the trains, the motor-cars, the buses in the streets, the offices, the schools and the shops were crowded with people indifferent to the danger. When the parachute was about 2000 feet above them the burden suddenly exploded. About the centre of the explosion there appeared a blinding fiery ball of terrific heat, and an explosive blast commencing at nearly 1000 miles per hour swept the city, crushing all in its path for miles. Of the 300,000 inhabitants, 90,000 were killed and 100,000 were injured. Within minutes the world was made aware that the first atomic bomb had devastated the delta of Hiroshima.

Later some said that the bomb had missed its target, the great naval dockyard at Kure, a few miles over the hills. But the bombing was accurate. Hiroshima was the main embarkation port for Japanese troops proceeding overseas.

What actually had happened?

I do not wish, nor am I able, to give you a learned dissertation on the chemistry and the physics of an atomic bomb; but something should be said in explanation, and I will briefly review the history of explosives and their application to missiles.

The Chinese would seem to have devised gunpowder, which the Moors, centuries later, introduced into Europe. This innovation sealed the doom of castles as defences. The English used the explosive for the first time in their weapons at Crecy in the fourteenth century, and until the nineteenth century little if any advance was made. In 1866 Alfred Nobel of Stockholm invented dynamite, a nitroglycerine product. Nobel was a man of high ideals; his purpose was the driving of rails and roads through

[1] Read at a meeting of the Victorian Branch of the British Medical Association on June 15, 1955.

mountains, to facilitate intercommunication and understanding between peoples and countries. Others not so peacefully inclined realized the power of the weapon that he had innocently forged, and so modern missiles were developed, culminating in the ten-ton "block busters" and incendiaries that devastated European cities in World War II. (In one hour's bombing 40,000 people were wiped out in Dresden.)

Nobel was deeply disturbed at what he considered the perversion of his conception, and when he died in 1890 his considerable fortune was willed to the founding of international prizes named in his memory, for advances in physics, chemistry, literature, medicine, idealism and contributions towards world peace. The first Nobel Peace Prize was awarded to Henri Dunant, the founder of Red Cross.

When we were very young physics and chemistry were easy. The molecule was a combination of various atoms —the atom was the ultimate indivisible entity. In our experiments we played with molecules, disturbing their atoms with visual, auditory, olfactory or sometimes painful results—and these were all that the explosive experts applied until 0815 hours on that fatal August 6, 1945.

Many years ago the Cavendish Laboratory, inspired by that great British chemist Lord Rutherford, had demonstrated that the atom is not the ultimate irreducible unit, but contains a system similar to a miniature solar system —a central sun or nucleus, a region of enormous electrical forces and various planet particles or electrons held by lesser electrical forces. These forces vary within each element.

If by any means this nuclear system could be disrupted, the atom would fall asunder with the liberation of these enormous electrical forces. This disruption or fission was achieved by a German chemical physicist, Otto Hahn, in 1939.

Had World War II not supervened, by now the world may well have been blest with the mastery of this new source of energy in industry, for as Professor Falls of Oxford has reminded us, scientific discovery and advancement have not only been applied to warfare, but in many instances have been due to the demands of war, as exemplified in the influence of war on the progress in surgery. When atomic energy is harnessed to the service of man, as appears likely in the near future, it must be remembered that this was developed in order to bring about his destruction, for the essence of war is violence, and instead of beneficence the atom bomb burst over Hiroshima and Nagasaki. The flash blinded those thousands, the heat burnt and shrivelled them, the blast crushed and buried them beneath the shattered structures, and the radiation destroyed the cells of their vital organs; so, blinded, they died a triple death.

But there was nothing fundamentally new in the dangers of that atomic bomb. Man has always faced the blinding hazard of the sun's brilliance aud been subjected to the danger of heat since—to crown his emergence from the lower animals—he learnt to kindle a fire. Since the dawn of the earth's surface the blast of the elements has been his lot. When Krakatoa in the Sunda Straits erupted on August 27, 1883, the surge went round the world four times before its force was spent. Since the beginning of time irradiating cosmic particles originating in interstellar space probably from nuclear excitations have fallen from the heavens above, and since radioactive elements have existed in the earth man has been subjected to radiation from beneath. With the increasing therapeutic and diagnostic application of radium and X rays, individuals are being increasingly submitted to radiation upon the earth's surface. But never before 0815 hours on August 6, 1945, had mankind been involved in such a catastrophic combination—such a simultaneous multiple of all these terrible forces.

There are now atomic bombs of various sizes and powers; those dropped over Hiroshima and Nagasaki are known as nominal bombs which, in energy released, are each equivalent to 20,000 tons of trinitrotoluene. Let us consider the various manifestations of the sudden liberation of this enormous energy from a nominal bomb exploded 2000 feet above ground, on a clear, still day, as meteorological conditions are factors in the effects.

The flash of light is more than ten times the brilliance of the sun as received on the earth's surface. In an experimental explosion in 1951 just after dawn, the glare was visible up to 400 miles from the explosion. The actual flash, if witnessed, will produce at least temporary blindness by exhaustion of the visual purple. The brilliance may disappear within seconds.

It is estimated that within one millisecond of the explosion, the ball of fire consists of particles heated to 300,000° C. This heat is radiated first in a pulse of ultraviolet wave moving with the velocity of light outwards in straight lines, and within a few seconds in a more damaging infra-red wave. In the passage through the air, laden with various particles, this heat is rapidly reduced; but up to about one mile from ground zero (the point immediately beneath the burst), wood is ignited and individuals directly exposed burnt to death. Severe burns will result in a distance up to two miles and mild heat injury beyond that distance, and warmth may be felt up to ten miles. At Nagasaki burns were recorded nearly eight miles from ground zero. Added to this primary hazard is the destruction by secondary fires.

The expansion of gases resulting from any explosion initiates a wave of pressure known as blast. Many of you have experienced this sudden pressure from gun-fire. Some of you have even suffered a ruptured ear drum from this force; but unless you have seen the effects of the blast, even from a nominal bomb, in the words of our allies "You ain't seen nothing yet". We have read and seen pictures of the havoc wrought by a hurricane or may even have experienced it. A terrific hurricane is a wind of probably not more than 150 miles per hour. The cyclone that devastated areas in southern Queensland earlier this year was travelling at 120 miles per hour. The blast or shock wave from a nominal bomb commences to travel at the rate of at least 800 miles per hour; this rate is reduced to about 200 miles per hour one mile from ground zero, but is still driving at 50 miles per hour over a diameter of five miles. While the human body can resist these sudden short but extremely high pressures, it cannot withstand the injury of steel and masonry and other debris hurled and flung about by the giant hand of this devastating force. In Japan it is estimated that 85% of the injuries from the bomb were the results of heat and blast.

Any electric discharge will result in the emanation of certain invisible but powerful particles and rays. Fortunately, in an air burst the most powerful are the least likely to penetrate human tissues at ground level. In a high air burst we need consider only those of short-wave γ rays. If these rays penetrate the human body, certain changes inevitably follow. The effects are produced by an ionizing action within the penetrated cells—a liberation of oxygen and a consequent oxidation process. If this is adequate to affect the cell nucleus, the cell dies. If sufficient vital cells are killed, the individual dies.

So now we know what happened at Hiroshima and Nagasaki: injury by brilliant light—injury by great heat —injury by objects blasted about—and injury by damaging rays.

The first three of these damaging agents present no new medical problems, although as each is complicated by radiation, each hazard is thereby enhanced. The radiation hazard, while undoubtedly of least importance in Hiroshima and Nagasaki, has captivated popular interest, and because of ignorance, popular fear. The presence and the power of γ rays are readily detectable and measurable by simple means even before any apparent damage has resulted. The unit of this power is the Röntgen, or simply the r.

It has been explained that the effect on the body tissues is the result of an ionizing action within the cells, and certain cells are more susceptible than others to this damage, generally in the following order: (i) lymphocytes and lymphatic structures; (ii) the myelocytic series; (iii)

epithelial structures—secreting glands, testes, ovaries, skin, gastro-intestinal tract, pulmonary alveoli, renal tubules; (iv) endothelium, blood vessels, heart; (v) connective tissue; (vi) muscle cells; (vii) bone cells; (viii) central nervous system. Cells are more susceptible to injury during activity or mitosis, and this fact, as you know, is applied in controlled deep X-ray therapy for certain pathological conditions. Several other factors determine the effects of radiation; but the differing sensitivity to radiation demonstrated by different species indicates the risk of error in applying to human physiology the results of animal tests. Moreover, there is a varying sensitivity among individuals of the same species. The duration of exposure and the area of exposure are of prime importance. A man may continue to enjoy one or two whiskies a day for years; but if he takes a bottle at one sitting the enjoyment is transitory. Radiologists and their technicians may be penetrated by 0·3*r* each week of their working lives with no ill effects; but if 100*r* came their way in one uncontrolled general application some sickness would probably follow. The single application of more than 100*r* may be necessary in taking an X-ray film of a frontal sinus, and more than 4000*r* in one dose may be necessary in radiotherapy; but if all other parts are adequately shielded from these exposures, no serious ill effects will ensue.

On the experience in Hiroshima and Nagasaki after the air burst of nominal bombs, the effects of total body exposure may be grouped as follows according to the dosage received: group I: subjects receiving radiation beyond the median lethal dose (500*r*); group II: those receiving 400 to 500*r*; group III: those receiving less than 400*r*.

Most of those in group I will die. Within hours of exposure there is increasing weakness, with thirst, nausea, vomiting and diarrhœa. Those who have received the heaviest doses rapidly become exhausted and die within two or three days. Others may appear to improve; but after a latent period of three or four days these symptoms reappear in an increasingly severe degree, accompanied by steplike changes in temperature rising to hyperpyrexia until death ensues ten to twelve days after exposure.

Subjects in group II live long enough to show blood tissue changes, and 50% survive. The same symptoms appear somewhat later—weakness, thirst, nausea, vomiting and diarrhœa—with improvement after three or four days, often sufficient to permit return to duty. After eight to ten days of this remission the symptoms recur; epilation begins at the end of the second week, buccal ulceration, bloody diarrhœa, hæmorrhages and petechiæ follow, and later aplastic anæmia. Of these subjects, 50% die of infection, the others slowly recover over months.

Of group III subjects, all who have received 200 to 400*r* will be ill, but very few will die; of those who have received 100 to 200*r*, most will be ill; of those who have received less than 100*r*, a few will be ill.

It is to be noted that the germinal cells of the ovary and testis are among those more susceptible to radiation, and considerable inquiry is being made as to the later results of this susceptibility in those who have recovered after exposure to atomic radiation. In view of the complexity of the fate of genes, many generations may need to be surveyed before any firm pronouncement can be made; but very little is known at present regarding the quantities of ionizing radiation required to produce genetic damage in man. For many years research with fruit flies and later with mice, because of the rapidity of their reproduction, has demonstrated mutation changes produced by radiation, and some workers go so far as to sound a strong note of warning against the increasing use of diagnostic X rays. One physicist has said: "I feel there may be less risk from tuberculosis than from mass X-raying." Many alarming, grotesque, and so far scientifically unfounded statements are made concerning this genetic hazard. These statements are generally made either by ignorant people who always fear the worst, or by people untrained in the science of genetics who do not hesitate to make a spectacular and flamboyant appearance in this field. Within twelve months of the atom bomb explosion over Hiroshima 25,000 babies were born in the area; apparently there has been among them no unusual incidence of abnormalities over the last nine years with the possible exception of chronic leuchæmia, but in the absence of adequate controls this is doubtful. However, there is some evidence of an abnormally high incidence of this condition among the radiological workers in certain countries.

So far we have considered the effects of a nominal bomb exploding about 2000 feet above ground.

If such a bomb was to burst on the ground or under the sea, the particles immediately in contact with the discharge would differ very materially from the particles normally in the air, and their reception and retention of radioactivity would present a new problem. While the blinding, burning and blasting effects may be modified and lessened, by the up-surge of irradiated particles from the ground material or water and the persistence of their radioactivity, this danger is greatly enhanced. Down wind these particles, before they decay, fall out over a large area while still —to a degree dependent on their composition—persistently radioactive, contaminating air, skin, food and drink within their path.

Such is the advance in civilization that we have passed far ahead of the fission atom-bomb of 1945 and now have produced a bomb which, when exploded by fusion, emits a flash of heat, a blast, many times more powerful than those which devastated Hiroshima, with a consequently more dangerous fall-out. This is the hydrogen bomb, and even more powerful weapons are contemplated.

This prospect tends to send our imaginations into panic or to numb us into hopeless apathy. But these extremes get us just nowhere.

Certainly the calamity of an atom-bomb explosion will inevitably mean great loss of life and widespread destruction—the hydrogen bomb more so, to a terrifying degree. But we can do something, firstly to prevent this catastrophe, and secondly to minimize the effects.

In our lifetime the decent nations have twice been involved in a world war for which none of them was really prepared. Whatever our walk of life, we always remain a citizen, with the privileges and the responsibilities which this implies. Let us try to avoid the third world war by being totally prepared. Quite possibly the British development of dreadful new war gases may have held back our enemies from loosing this horror in World War II; so may it be if we push on with a stock of just a little better atom-bombs, which we have done. At the same time, let us all honestly do our humble best to remove the mistrusts, the misunderstandings, the greeds, the jealousies among the nations that lead to war. There is just a chance that this may succeed, and if so the world will be spared the unbelievable, unimaginable horror of atomic warfare. But our sincere hopes may not be realized, and once again the dogs of war may be unleashed. If that happens, atomic warfare is with us, everyone of us—and that means with you and with me and with the millions of us who make this community. Atomic warfare is now conventional warfare. Some may be in uniform—they may be the lucky ones; many more will be civilians.

Mr. Richard L. Meiling, Director of Medical Services to the Secretary for Defence in the United States of America, reminded us in 1950 that "military medicine is an integral part of civil medicine—essential in every physician today as were the studies in anatomy, physiology and pathology during his medical school days". Let us therefore consider the medical problems presented by the various traumatic agents resulting from an atomic explosion.

We need not be concerned with the temporary blindness resulting from the exhaustion of the visual purple through flash; if the victim survives, his vision returns. Among certain of those who suffered at Hiroshima there are still seen opacities, either in the lens or in the posterior chamber of the eye, possibly the result of heat. Untreated, these slowly clear and disappear, as has been observed by the Atom Bomb Commission studying the late effects of the bombing of Hiroshima.

Heat presents the great problem of massed burns. Many and various basic principles in the treatment of burns have been advocated during our medical lifetime; but surely by now we can determine and teach certain standard and efficient principles. How many in our own community are soundly informed on the principles of first aid to burns? The answer to massed burns is, firstly, a determination of sound and standardized first-aid principles and the earnest promulgation of these among all the responsible members of our community. Sound and standardized methods of treating burns should be taught in all our medical and nursing schools. Medical students and nurses are taught so many different ways to approach their problems that they often have great difficulty in determining a precise plan when faced with an emergency. Adequate equipment, including dressings, blood and blood substitutes, together with transfusion apparatus, must be available.

Blast presents the problems of massed and varied injuries. Here again, simple standardized principles in first aid and in skilled treatment are necessary. These are more standardized than those concerning burns. Again, adequate facilities for such treatment must be assured.

Just as the injuries from heat and blast may be reduced or avoided by adequate shelter, so may the effect of nuclear radiation be minimized and γ rays delayed or diverted by adequate protection. It may be accepted that the median lethal dose of γ rays received over the whole body is 500*r*, and much higher doses may be reduced to this or lower levels by suitable screenings, in which one inch thickness of lead is equivalent to two inches of iron, six inches of concrete or eight inches of packed soil.

A good rough rule is that 20 inches of tightly packed soil will reduce the γ flash by a factor of six—600*r* would become 100*r*.

In the case of fall-out, the energy of the γ rays is considerably less than that of the γ flash, and the reduction factor here is 100—600*r* would be reduced to 6*r*. Simple scaling laws can provide similar information concerning protection from various doses.

Considerable laboratory research has been devoted to other means of reducing the effects of γ rays. Shielding of the spleen has lessened the effects of whole-body radiation in mice. If given up to within two days of exposure, splenic extracts and bone marrow injections have alleviated the effects in animals. If given within one hour of whole-body exposure, cystine, thiamine and glutathione, by limiting the available oxygen, have proved of value in mice. The parenteral administration of cortisone has appeared to contribute to reduction of the initial inflammatory reaction in guinea-pigs. I understand that pyridoxine given by mouth is used by radiotherapists to diminish radiation sickness by its action on the liver, that chlorpromazine by injection is useful in controlling the vomiting. Certain components of the vitamin *B* complex may build up a resistance, and it will hearten you to hear that alcohol taken internally appears to limit the effects of radiation sickness.

All these measures may ultimately prove of value; but in the light of our present knowledge we must rely mainly on education and adequate protection, then on rest and nursing, on the administration of antibiotics (not sulphonamides), on the maintenance of nutrition and on fluid and electrolyte balance, and on fresh whole blood transfusion when indicated.

No longer, therefore, are we justified in being sent into panic or numbed into an apathetic hopelessness. Certainly I have presented the effects only of the nominal bomb; but the horrible multiple of these resulting from a hydrogen bomb should not deter us from the effort of facing them.

I have not touched on the terrible medical problems resulting from the inevitable loss of the lives of many trained medical and nursing personnel, or on the inevitable destruction of hospitals, medical equipment, transport, communications, fire-fighting equipment, food and water, and the dreadful effects of panic. In Hiroshima 90% of the doctors, 95% of the nurses and 85% of the firemen on duty were immediate casualties. These problems must be faced and plans prepared to meet them.

I believe that at present not one hospital in the whole of Australia could cope adequately with the simultaneous admission of 20 severely burnt patients and continue to discharge the regular hospital commitments. But an atomic explosion on a city would involve not only 20 or 200 or 2000 severe individual burns, but many more. This presents a national problem.

Never again should the cry go out, as it went from those poor destitute and bewildered people of Hiroshima and Nagasaki on those dreadful days ten years ago, "Nobody told us!"

There is a deal of factual information available concerning atomic and thermo-nuclear weapons. Much of this information is available—rightly—for anyone who seeks it. Colonel W. D. Refshauge, O.B.E., has been most active in collecting and surveying this information, and I am extremely grateful to him for the sifting of much of the material that is here presented.

Finally, let me remind you of the quotations that began this lecture:

> Only the Devil could have devised this Godless weapon. Mankind can never withstand the blinding flash the seering ball. Its invention means the end of humane nobility.

This dates back to the fourteenth century, after the English soldiers under Edward III first used gunpowder at Crecy on August 26, 1346.

> Prompted by widespread fears that this new weapon of mass destruction might wipe out Western civilisation the Pope today issued a Bull forbidding their use by any Christian state against another whatsoever the provocation.

This dates back even further—to the twelfth century, when that deadly weapon the cross-bow was introduced into warfare.

UROLOGICAL CONDITIONS IN PREGNANCY.[1]

By A. S. B. STUDDY,
Sydney.

I PROPOSE to deal with some of the more common urinary tract complications of pregnancy and not to attempt any systematic classification.

Hæmaturia.

Hæmaturia usually occurs in the later stage of pregnancy, seldom before the fifth month, and varies from gross bleeding to microscopic amounts of blood. The hæmorrhage is usually painless, sometimes profuse and generally recurrent. It may clear up in the puerperium without there being any discoverable cause. It may be due to any of the following abnormalities: (i) pyelonephritis, (ii) tuberculosis, (iii) hydronephrosis, (iv) polycystic disease, (v) neoplasm, (vi) chronic nephritis. Urinary calculi do not often cause hæmaturia, as dilatation of the upper urinary passages is present.

Increased vascularity or ruptured varices in the upper part of the urinary tract may sometimes cause hæmaturia.

Finally, hæmaturia in pregnancy should always be investigated.

Bacteriuria.

The presence of bacteria and pus in the urine and symptoms of urinary tract infection confirm the diagnosis of pyelonephritis. The causal organisms are usually *Bacterium coli*, staphylococci and streptococci in that order. The presence of bacteria in the urine without pus cells may be of no significance.

Bacteriuria is estimated to occur in about 11% of pregnant women. Infection may occur by one of the

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on May 26, 1955.

following three routes: (i) by the blood-stream—for example, from some septic focus; (ii) from the ascending colon by lymphatic spread to the kidney; (iii) by an ascending infection from the bladder. Probably the most important predisposing cause of pyelitis is urinary stasis in pregnancy.

Dilatation and kinking of the ureter and dilatation of the renal pelvis and calyces are readily demonstrable in pyelograms. This, of course, is more pronounced on the right side; it may be attributable to the fact that the uterus commonly lies slightly over to the right side and causes pressure on the right ureter, and also to the fact that the left ureter is protected by the sigmoid colon. I understand that Dr. Gee is discussing this subject in more detail.

Urolithiasis.

Urolithiasis is not a very common complication of pregnancy. On looking through the records at the Royal North Shore Hospital of Sydney I can find only two cases recorded in the past three years, though other cases may, of course, have passed unnoticed. The histories of these cases were as follows:

The patient, aged thirty-eight years, was pregnant for the third time; her other children were aged six years and fourteen months. Her last menstrual period had occurred in July, 1953, and her expected date of confinement was in April, 1954. On December 14, 1953, an X-ray examination revealed a large calculus in the uppermost calyx of the right kidney. Both kidneys were functioning normally, and the course and calibre of both ureters were normal. The patient was admitted to hospital on April 23, 1954, with hypertension (blood pressure 150 millimetres of mercury, systolic, and 90 millimetres, diastolic), and discharged on April 30. She was readmitted to hospital on May 14, and had medical induction followed by normal labour and delivery of a living female infant. On May 20 microscopic examination of the urine revealed 15 pus cells per high-power field, and culture produced a growth of *Bact. coli.*

The patient, aged forty-four years, was pregnant for the sixth time. Her last menstrual period had occurred on July 8, 1951. Her expected date of confinement was April 19, 1952. Investigation of her previous history showed that a renal calculus had been removed from her left kidney. X-ray examination revealed multiple calculi in the right kidney. The patient was delivered of a living male infant on April 12; labour and the puerperium were normal. Her blood pressure was 140 millimetres of mercury, systolic, and 100 millimetres, diastolic.

In pregnancy, calculi are much more prone to be associated with infection, progressing to pyelonephritis and pyonephrosis. In early pregnancy, before dilatation and atony of the upper urinary passages occur, pain is more likely to be a prominent feature, especially when a calculus is found in the ureter or at the pelvi-ureteric junction. Later on in pregnancy, the presence of an infection resistant to treatment may lead to an investigation by excretion pyelography, the calculus being revealed. Fœtal parts may obscure the picture and necessitate the taking of retrograde pyelograms (oblique and lateral views). Treatment will depend on the stage of the pregnancy, the severity of the symptoms, the degree of infection and the size of the calculus. It should include the administration of suitable antibiotics and an adequate daily fluid intake of at least three or four litres.

If surgical intervention becomes necessary because of the persistent infection and obstruction due to the calculus, percystoscopic removal may be attempted. Failing this, pyelolithotomy or ureteric lithotomy may be necessary.

In the later months of pregnancy when surgical interference becomes difficult, nephrostomy may temporarily relieve the symptoms, until a more radical operation can be performed later.

Obstruction is the main factor to be considered in urolithiasis. If there is no obstruction, the calculus may be left until the pregnancy has been completed, and then removed at a suitable and convenient time.

Hydronephrosis.

There are many degrees of hydronephrosis that must be taken into account when the advisability of attempting or continuing pregnancy is being considered. Attention should also be given to whether the condition is unilateral or bilateral, and whether infection is present. The physiological dilatation of the urinary tract during pregnancy would tend to increase the extent of the hydronephrosis, and infection is not unlikely to occur. The decision as to whether pregnancy should be embarked upon by a patient suffering from hydronephrosis will depend on the degree and cause of the hydronephrosis, on the presence or otherwise of infection, and on whether the renal function is normal. If the hydronephrosis is due to aberrant vessels, it will not be influenced by pregnancy, but if it is due to intrinsic ureteric stricture, it will be greatly influenced by pregnancy.

More severe degrees of hydronephrosis should be treated surgically before pregnancy is attempted. If pregnancy occurs and infection develops and renal function deteriorates, it may be necessary to terminate the pregnancy. Prior to termination of pregnancy on account of infection with hydronephrosis, it is often worthwhile trying a peristaltic stimulant such as eserine (the administration of which must be continued till the end of the puerperium at least). Failing that, indwelling ureteric catheters may be tried. Further pregnancy should not be attempted until the condition has been corrected.

Tuberculosis.

Tuberculosis is a rare complication of pregnancy; it is always secondary to tuberculosis elsewhere, and never a primary infection in the kidneys. Pyuria, acid urine, and absence of growth on attempted culture from the urine should direct attention to the possibility of tuberculous infection, and guinea-pig inoculation should be tried as well as excretion pyelography. Once tuberculosis is diagnosed, the decision to continue or terminate pregnancy will depend on whether the disease is unilateral or bilateral, on the duration of pregnancy and on the severity of the symptoms.

In early pregnancy, with unilateral infection, nephrectomy can be performed and pregnancy allowed to continue in very exceptional cases. Streptomycin and PAS will not adversely affect the fœtus.

In the later months of pregnancy when the child is viable, pregnancy may be terminated after the adoption of suitable chemotherapy, and nephrectomy may be performed later if necessary.

With bilateral renal tuberculosis termination of the pregnancy should be performed immediately.

Tuberculosis, if discovered early in pregnancy and shown to be rapidly advancing as demonstrated by pyelography, is a definite indication for termination of pregnancy.

Incarcerated Uterus and Urinary Retention.

As a general rule the third month of pregnancy has almost been completed, and the uterus has become slightly incarcerated, before any symptoms develop.

It is the bladder which is primarily affected in retrodisplacement of the gravid uterus, and the first evidence of the displacement is in most cases frequency and difficulty in urination.

Indeed, so characteristic of the condition is this symptom that dysuria in the early months of a pregnancy should always arouse in one's mind the suspicion of backward displacement. Before long, retention of urine develops and subsequently overflow incontinence. When this stage is reached there is considerable tenderness to pressure and distension of the lower part of the abdomen, and the patient makes complaint of pain and discomfort in that region. The time of its onset is, as a rule, the twelfth to the fourteenth week.

With retroversion of the second degree and retroflexion difficulties in urination appear earlier than with extreme retroversion.

In third degree retroversion, the fourteenth or fifteenth week may be reached before symptoms develop.

Several theories have been advanced to account for this retention of urine. Œdema of the bladder wall, produced by the cervix pressing upon the veins and retarding the circulation about the neck of the bladder, is the initial local change. Rarely, sufficient pressure is exerted against the urethra to cause obstruction with urinary retention. Myoma in a pregnant uterus may be a factor in some cases. Sacculation of the uterus may relieve the pressure within the pelvis.

In the treatment of this condition, the bladder should first be decompressed with the passage of a fine catheter; after that reposition may be attempted with the patient in the knee-chest position, and if this is not successful, an anæsthetic should be given.

Stress Incontinence of Urine.

Stress incontinence of urine, a troublesome complaint, is by no means uncommon after labour, and is usually associated with a cystocele. This, of course, is caused by over-stretching of the supports of the anterior vaginal wall, especially in the region of the bladder neck. Prevention consists of ensuring that the bladder is maintained empty during labour, and that a catheter is passed before application of forceps.

Early episiotomy, when necessary, will materially assist in preventing this complication.

The symptoms usually progressively decrease over two or three months, and a cure can be hastened by the regular performance of "perineal exercises". Failing this, a modified colporrhaphy may be tried, and finally a urethro-vesical suspension of the Marshall Marchetti type may be necessary.

Vesico-Vaginal Fistula.

Vesico-vaginal fistula is fortunately nowadays a rare complication. It may be caused by (*a*) prolonged compression of the structures between the fœtal head and the mother's pubes or (*b*) operative manipulation, which may produce a tearing of the vesico-vaginal septum. Spontaneous closure may occur, and is assisted by an indwelling catheter.

After previous vaginal plastic operations for urinary stress incontinence or vesico-vaginal fistula, each case must be carefully evaluated before the patient is submitted to vaginal delivery. Vaginal delivery displaces the bladder neck downwards and forwards from its its normal attachments, and the condition may recur.

Post-partum Atony and Retention of Urine.

Post-partum atony and retention of urine are seen infrequently with better nursing and with confinements in hospital.

Relieved suddenly from the increased intraabdominal pressure by the delivery of the child, the kidneys become active. In the third stage of labour it is not rare to find the bladder full or even over-filled, which makes an obstacle to the delivery of the placenta.

In the first twelve hours not infrequently retention of urine is present, which sometimes may be pronounced. This may be due to lack of elasticity of the bladder, inability to urinate in the horizontal position, the swelling of the vulva and urethra, kinking of the urethra, reflex spasm of the sphincter from stitches in the perineum, and injury to the bladder trigone and urethral orifice with œdema.

If retention of urine progresses to bladder atony, an indwelling catheter left in position for some three or four days will usually result in a cure.

Catheterization for residual urine will, of course, be necessary until the bladder is functioning normally.

Pregnancy after Nephrectomy.

After nephrectomy, the state of the remaining kidney and the pathological condition which made it necessary to remove the other kidney will determine whether the pregnancy should be allowed to continue or not.

Pregnancy should not have any adverse effect on the remaining healthy kidney. If complications do ensue, it is more serious when the right kidney remains, as dilatation and stasis are more pronounced on the right side. Before pregnancy is attempted, or if pregnancy has begun, before it is allowed to continue a microscopic and cultural examination of a catheter specimen of urine should be performed, as well as excretion pyelography and renal function tests. Stasis in the upper part of the urinary tract may interfere with the true assessment of renal function in the later stages of pregnancy, and a urea clearance test should be carried out as well.

Malignant disease in the kidney previously removed is a relative contraindication to further pregnancy, for hypernephroma is rarely bilateral.

Tuberculosis is a more doubtful contraindication, and some authorities consider that pregnancy should be allowed after three years if the patient has made a complete recovery. In the case of a patient already pregnant when first examined, if there is evidence of impaired renal function, pregnancy should be terminated. If infection is present which responds rapidly to chemotherapy and the pyelogram is normal, pregnancy may be allowed to continue under close supervision.

Congenital Polycystic Kidneys.

The fact that the symptoms of congenital polycystic kidneys are unlikely to manifest themselves during the reproductive age group makes this condition a rare complication of pregnancy. It may become recognized in a diagnostic urogram for hæmaturia or urinary infection. Should renal function be satisfactory, pregnancy may be allowed to continue. Termination of pregnancy may be necessary if renal function is impaired or deteriorating, or if the symptoms cannot be controlled by ordinary means. (A blood urea level around 60 milligrammes per 100 millilitres may be satisfactory, but a level of 90 milligrammes per 100 millilitres may be dangerous.)

Pelvic Ectopic Kidney.

Pelvic ectopic kidney may give rise to no symptoms, and may become evident only on pelvic examination when a mass is palpated separate from the pregnant uterus. Backache and lower abdominal pain may be present. This condition has on occasions been erroneously diagnosed as an ovarian cyst. At times the kidney function may be impaired, and the kidney may become hydronephrotic or the seat of pyelonephritis. A symptomless ectopic kidney is no contraindication to pregnancy, nor does it warrant termination of pregnancy. In the event of its causing obstructed labour, Cæsarean section may be necessary. Usually the kidney is mobile enough to allow it to be pushed up during the descent of the head into the pelvis.

Anuria.

Anuria was formerly thought to be due to cortical necrosis, but now it is considered that it may be due to lower nephron nephrosis, such as occurs in burns, unmatched blood transfusions and crush injuries.

In obstetrics this may occur in septic abortions, post-partum hæmorrhage, toxæmia or accidental hæmorrhage. Tubular necrosis will, if prolonged, lead to cortical necrosis. The ætiological factors in anuria are as follows: (i) shock or hæmorrhage may be involved. Renal tissue is highly susceptible to anoxia, and circulatory renal insufficiency is a very common cause of anuria. Accidental hæmorrhage is by far the most frequent cause of cortical necrosis in obstetrics. (ii) Mismatched transfusion is another cause. This can be diagnosed by the finding of hæmoglobinuria or bilirubinuria. Anuria occurs in abortion with blood incompatibility. *Clostridium welchii* infection may cause anuria owing to its hæmolytic exotoxin. (iii) Anuria may be due to toxic necrosis—for example, mercuric poisoning, when a mercury preparation is used as an abortifacient. (iv) Anuria may be associated with sepsis.

On histological examination, in cortical necrosis all the elements of the nephron are destroyed, and necrosis and thrombosis of the arterioles are also present. In tubular necrosis, the glomerulus survives. Once the glomeruli have been grossly damaged, restitution is no longer possible; but with tubular damage as in acute tubular nephrosis, recovery is always possible in time. If death from uræmia can be prevented, most patients with anuria related to pregnancy should recover.

Patients with gross cortical necrosis die of uræmia no matter what treatment is adopted, as regeneration of glomeruli does not occur.

It is impossible to distinguish between anuria due to cortical necrosis and anuria due to tubular necrosis. Tubular necrosis may be divided into three stages, as follows. (i) The precipitating stage, usually lasting seven to ten days, or longer. This is characterized by a rising blood urea level and by retention of toxic metabolites due to total absence of renal secretion of any sort. (ii) The stage of oliguria or anuria, due to the glomerular filtrate's escaping directly through the denuded basement membrane and returning to the blood-stream. (iii) The stage of diuresis. The recovery of glomerular filtration with an absence of tubular function leads to the kidneys' excreting what is in fact a pure glomerular infiltrate. Clinically this results in electrolyte disturbances and dehydration. Potassium, chlorides, sodium, calcium and magnesium may be lost in abnormal amounts. Increase and variation in the specific gravity of the urine are evidence of returning tubular function.

Treatment is identical for both cortical and tubular necrosis, so that the diagnosis of the respective conditions is unimportant. Conservative treatment is best for both conditions. There is no place for decapsulation of the kidneys. Splanchnic block, if employed at all, must be used in the early stages; but its value is still doubtful.

In anuria it is important to avoid over-hydration.

Although electrolyte disturbances are significant in uræmia, they are of secondary importance to water imbalance. One litre per day will suffice to replace the fluid lost in sweat, by breathing and in the fæces. However, the body should not be overloaded with electrolytes such as sodium chloride, and the administration of the potassium ion in anuria is particularly dangerous. Potassium citrate, bottled fruit drinks and coffee should be avoided.

During the anuric stage, deficiency of sodium and chloride and other electrolytes does not appear to be harmful, but during the stage of diuresis it is important to correct imbalance. A diet of high carbohydrate content depresses endogenous nitrogen metabolism, and Boorst, in 1948, developed a diet along these lines. It consists of 400 grammes of glucose and 100 grammes of peanut oil made up in a litre solution. Tube feeding may be instituted, and the mixture is dripped at the steady rate of one litre in twenty-four hours. Vomitus is collected, filtered and returned to the stomach.

This discussion of the treatment of anuria has purposely been restricted in detail, as the assistance of a reliable biochemist is essential in dealing with this condition.

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UROLOGICAL COMPLICATIONS IN PREGNANCY.[1]

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EXCEPT for the cardio-vascular system, the urinary tract is the most vulnerable to the deleterious effect of pregnancy, and before passing to consider the complications encountered, I propose to review briefly the anatomical and functional changes concerned. These changes are brought about by two main factors—(*a*) the effect of the enlarging uterus, and (*b*) hormonal influences. The endocrine changes predominate, and it must be noted that they affect the body as a whole. The tone of smooth muscle including the bowel is lowered, and likewise its motility. At the same time there is an increased capacity of hollow viscera, such as the bladder, and a decrease in reactions to normal stimuli. The ureter undergoes hyperplasia of its musculature, particularly in its lower end, and its period of greatest diminution of tone is from the fourth to the eighth month. The tone then improves, to fall sharply after delivery, becoming finally normal again about two months later. Some dilatation is thus produced, to be greatly accentuated by the effects of the enlarging uterus. This in 80% of cases rotates to the right, causing pressure after the fourth month on the right ureter, which is more exposed at the pelvic brim than its opposite number. It will be recalled that the sigmoid colon protects the left ureter, which also lies more posteriorly on the left iliac artery than occurs on the right side. The pelvic part of the ureter does not show this dilatation, and on the left side is actually carried more anteriorly by the rotation to the right. *Primiparæ*, owing to better abdominal wall musculature, show more pronounced pressure effects than *multiparæ*, and quadripeds as an antithesis show none at all. These two factors thus combine to produce lowered tone and urinary stasis, upon which depend nearly our whole concept of renal tract complications. The bowel also suffers this lowered tone and pressure effects, and constipation to some extent usually occurs. Renal function is quite normal, as is shown by blood chemistry tests, and the apparent deficient excretion revealed by excretion urography or cystoscopy seems to be present only because of delayed conduction along the post-renal collecting system.

Infections of the Urinary Tract.

Infections predominate among the urinary complications in pregnancy, and when considered in terms of end results in later years, they are second only in importance to the toxæmias. Stasis in the upper urinary channels and bowel is the chief predisposing factor. The infection appears later in *multiparæ*, and enters via the urethra directly or from the bowel. From the latter it reaches the kidneys either by the blood-stream or by the lymphatics, which incidentally are more directly connected with the right kidney than the left.

Symptoms and Signs.

The symptoms and signs of pyelonephritis in pregnancy are so atypical that the diagnosis mainly rests on the presence in the urine of bacteria and pus, and on its differentiation from other fevers. It must also be borne in mind that the other common urinary tract diseases such as stone and hydronephrosis are in pregnancy so different from their usual symptomatology that they are readily masked by pyelonephritic manifestations.

Bacteriuria is more frequent in pregnant than in non-pregnant patients, and such patients should be carefully watched for minor ailments such as acute colds, chills or tiredness, which often mean a flare-up of the infection, with the appearance of pyuria. Only in the more severe cases do we find fever, backache and a raised pulse rate.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on May 26, 1955.

Acute urinary bladder symptoms are uncommon, and there is no correlation between the degree of dilatation and the presence of either pain or tenderness. Very large dilatations frequently produce few local symptoms, and those that occur are mainly on the right side. Pain is more likely where peristaltic activity is still present; but except in well-advanced cases, the picture is indefinite, often rather vague, and diagnosed only by vigilance and by examinations of the urine.

It is thus important that a careful history should be taken, and inquiries made as to past urinary troubles. Even if these investigations give negative results, a catheter specimen of urine should always be examined microscopically and culturally examined for organisms. In this way asymptomatic patients with infection can be recognized, and prophylactic measures taken.

Prophylactic Measures.

Prophylactic measures consist essentially of attention to general hygiene, as we cannot modify the fundamental changes in the urinary tract which play so important a part, and we must recall that similar changes occur in other organs, such as the bowel. Therefore a routine of bowel regularity should be established early, and measures taken to prevent bowel stasis or constipation. This can be helped by a suitably proportioned diet, which should also be checked to make sure that it will not adversely affect any anæmia. The teeth should be examined for sepsis, and increased fluids taken as a routine measure—up to three litres daily. Frequent rest periods are also of great help in permitting better renal drainage, as this has been shown to be impaired in the upright position.

The sulphonamide drugs are our great stand-bys, and can be given in several weekly courses or over a long period, the usual dose being two grammes daily. If the patient is febrile, she should be put to bed until at least one week after her symptoms have subsided. If it is possible the prone position is best for drainage, as sitting up forces the kidney downwards. At times it helps if the patient lies on the opposite side to that affected, to allow the uterus to fall away from the ureter. Many question the efficiency of this measure, but I have been convinced of its value in several cases. The knee-chest position would of course be better, but is impracticable. Fluid intake must be pushed to the four-litre level, and the urine alkalinized. I prefer sodium bicarbonate for this purpose. Sulphonamide drugs should be given, and if necessary an antibiotic, according to the sensitivity of the organism.

If the obstructive element is not great, eserine sulphate (1/100 grain three or four times daily) will increase ureteric tone and help drainage, and should be used more than it is.

Catheterization of the ureters is now rarely necessary to drain the pelves. If the catheter can pass any ureteric kinks, it can give immediate relief of symptoms; but of course this is limited in its duration, and recurrence of symptoms is likely.

If infection does not speedily clear, or if it recurs, an excretion urogram should always be taken. I should like to stress this statement. The first four months is the period in which remedial measures can be readily undertaken, but too often this investigation is delayed. Some surprising conditions reveal themselves as a cause of continuance of pyelonephritic symptoms.

Mrs. A., aged thirty years, was three months pregnant when admitted to hospital for investigation of persistent pyuria with hæmaturia. The urine contained numerous pus cells and a heavy growth of *Bacillus coli communis*. Her blood pressure was slightly raised, and she was tender in both renal angles, and looked toxæmic. She had lost two and a half stone in weight in the previous eighteen months, and complained of lower abdominal pain, worse with movement. Investigation of her past history showed that she had had repeated attacks of pyelonephritis and cystitis since the age of fifteen years; attacks had occurred during her previous three pregnancies, eight, six and three years earlier respectively. A plain X-ray film revealed a large opacity in the bladder area. An indwelling urethral catheter was inserted and continuous drainage was carried out, and "Sulphaco" (triple sulphonamide) tablets were administered. After ten days her urine was sterile, though pus was still present, and excretion urography located the opaque area as a bladder stone. This was confirmed by a cystoscopic examination, and a cystotomy was performed to remove the stone. She was discharged from hospital ten days later, and her pregnancy continued uneventfully to term.

It should be noted that no investigations had been made during any of this patient's previous confinements.

Mrs. B. was admitted to hospital for investigation of persistent urinary infection and a raised blood pressure (to 150 millimetres of mercury, systolic, and 100 millimetres, diastolic). There had been three previous pregnancies, eighteen, seventeen and nine years before, and during the last she had had hypertension with left renal symptoms. Excretion urography revealed a mulberry stone in the left renal pelvis with back-pressure calyceal dilatation. She was then four months pregnant, so operation was undertaken and the stone removed. Her blood pressure returned to normal, the infection cleared, and she carried on without further trouble.

I think without doubt that this stone had been present during the previous confinement, and the patient had had persistent attacks of pain ever since then.

Calculous Disease in Pregnancy.

In my experience the commonest factor causing persistent infection is calculous disease in some part of the urinary tract. This is usually said to be of rare occurrence. Crabtree (1934) found it but four times in a five-year period at the Boston Lying-in Hospital, while Baird (1936) found it but ten times over a six-year period at the Glasgow Infirmary. Neither had ever seen a bladder calculus. However, at the Women's Hospital over the past five years, there have been at least 20 cases to my knowledge. I believe that this high figure is due to the alertness of the obstetrical staff in instituting a full investigation into any persistent infections or unexplained hypertension. Over the past two years the incidence has been once in every 600 confinements. In general, calculous pyelonephritis is not considered a factor in hypertension; but, although my records are not complete, four of my patients had a resting blood pressure above 150 millimetres of mercury, systolic, and 100 millimetres, diastolic, and in all it has returned to normal since operation.

Mrs. C., aged forty-one years, had had three previous pregnancies, but no history of them was available, and there were language difficulties. She was first examined at the Women's Hospital when four months pregnant, having been admitted for investigation of hypertension, albuminuria and pyuria. Excretion urography revealed a left renal calculus with back-pressure calyceal dilatation, and pyelolithotomy was performed soon afterwards. Her blood pressure returned to normal, her urine cleared and she had no further trouble. She has since undertaken a further pregnancy without complications, and was very well when examined recently, nearly five years after her operation.

Mrs. D., aged twenty-six years, was examined at the Women's Hospital in February, 1950, after her pregnancy had been terminated at three months. She had had three previous pregnancies, for which she had been confined elsewhere, the last two, three and two years previously, being characterized by hypertension, pyuria and albuminuria. Pus was found in her urine, proved cystoscopically to be coming from the right kidney and bladder only, and coliform organisms were grown on culture. X-ray examination revealed a right renal calculus which was duly removed, and seven months later she was symptom-free, with clear urine.

Mrs. E. was an interesting patient on whom I was nearly forced to perform a nephrostomy or pyelostomy near term. She had had repeated attacks of right-sided pyelonephritis and pain with raised temperature, but her urine was clear. There was considerable tenderness over the right kidney, and a plain X-ray film showed a probable calculus in that area. At a cystoscopic examination, nothing could be passed beyond the upper end of the right ureter, and no urine was obtained. Clinically, the patient had a pyonephrosis, but fortunately she came into labour the next day. An excretion urogram was then taken, confirming the lack of excretion from the right kidney, and three days later a pyelolithotomy with division of an aberrant vessel was performed. Pure pus drained from the kidney. A follow-up investigation three months later gave a surprisingly normal result.

In general it can be taken that calyceal stones are less harmful than pelvic stones, and that small stones which may obstruct the pelvis or ureter are more dangerous than large stones. Ureteric stones cause most trouble in the first four months when tone is good, and the symptomatology is as in the non-pregnant patient. From the fourth to the eighth month symptoms are less pronounced, but signs of infection more noticeable. Few stones are passed. During the last month and in the puerperium tone again increases, and calculi are frequently moved.

Mrs. F. had had attacks of right renal colic and was three and a half months pregnant. In spite of ureteric dilatations the calculus appeared to be firmly fixed in the pelvic part of the right ureter, and ureterolithotomy was performed. Pregnancy was then uneventful.

On the other hand, Mrs. G. was sent down from the country when seven months pregnant with a stone in the lower part of the right ureter. Because of the mechanical difficulties, and the absence of symptoms and back pressure with normal urine, conservative management was followed. Just on term right renal colic developed, and soon afterwards the patient came into labour. A further X-ray examination then showed the stone to have been passed, though it was never recovered. Three months later an excretion urogram showed only slight residual hydronephrosis.

The rule to be followed in the treatment of calculus in pregnancy is to remove all possible calculi in the first four months. In the latter half of pregnancy tremendous vascular changes complicate surgery, and there may not be time for strong wound healing. Open operation is usually undertaken only if there are major complications such as pyonephrosis.

Hydronephrosis in Pregnancy.

Apart from the urinary tract dilatations peculiar to the pregnant state, there are some patients who have a prior hydronephrosis, due mostly to congenital conditions such as aberrant vessels, strictures or hypoplasia. Some of these dilatations remain sterile, but many become infected and so give rise to persistent pyelonephritic symptoms. An excretion urogram makes the position clear, and treatment can be adopted accordingly.

Mrs. H., aged twenty-four years, was six months pregnant when first examined, having just recovered from two attacks of right pyelonephritis in rapid succession. An excretion urogram had revealed hydronephrosis. She was carried on with conservative treatment. A further attack occurred six weeks after confinement, and she complained of a constant loin ache six months later. A retrograde pyelogram now showed an upper ureteric obstruction, and at operation a stricture was found at the uretero-pelvic junction, which was surrounded with dense adhesions.

Mrs. I., aged thirty-one years, had a somewhat similar history during her confinement, with a heavy coliform infection. Later investigations again showed an upper ureteric obstruction, and at operation an aberrant renal artery was found to be the cause.

Mrs. J., aged thirty years, during her fifth pregnancy had recurrent left pyelonephritis with coliform organisms in the urine. An excretion urogram disclosed advanced hydronephrosis with a calculus in the lowest left calyx. Conservative treatment carried her through, but she continued to have symptoms after term, and a retrograde pyelogram revealed a congenital calyectasis, with heavy infection from that side. Nephrectomy was necessary.

Mrs. K. presented a similar history, but her pathological condition turned out to be a congenital hypoplastic kidney, with very little renal cortex. Such kidneys are frequently the site of chronic infection.

These cases thus show four different types of renal abnormality, uretero-pelvic junction stricture, aberrant vessel, congenital calyectasis and hypoplastic kidney, all of which produce urinary stasis with its liability to infection.

Other types of congenital abnormality that may occur are strictures at the lower end of the ureters, hydroureters opening into the bladder with golf-hole orifices, fused and malposed kidneys, and duplicated renal pelves and ureters. Such conditions are uncommon, but do occur and are readily diagnosed by intravenous pyelography.

Renal Cyst Disease.

Polycystic disease is occasionally encountered, but not often before the forties. Such patients may tend to hypertension, but usually present a problem only because of diminished renal function or pressure effects on a big kidney from an enlarged uterus—for example, bleeding and infection. Little treatment is possible, but I believe that these patients can often be helped *post partum* by Rovsing's operation. Solitary cysts do not as a rule cause trouble, but the following case is, I think, an exception.

Mrs. L. was examined after her pregnancy had been terminated at three months for hypertension, which was still present, though lowered. An excretion urogram revealed a tumour of the lower pole of the left kidney, which was thought after retrograde pyelography to be a cyst. This was confirmed at operation, but it was noted that the cyst was the size of a golf-ball, and entirely intrarenal, with a definite tense area of renal cortex forming its outer wall. The patient's preoperative blood pressure of 170 millimetres of mercury, systolic, and 110 millimetres, diastolic, dropped to normal by the end of the operation, was below normal for the next few days, and has been normal since—a duration of one month.

I believe it is possible that pressure of the cyst on this patient's renal tissue caused her trouble. Time alone will answer this.

I think that, from what has already been said, nobody can doubt the value of a urological investigation of any patient with recurring signs and symptoms. One further point I would like to make. An excretion urogram is said sometimes to show "old subinvoluted pyelitis of pregnancy". To me this often means that the ureter has become fixed with adhesions after a severe infection and periureteritis. This may take six months or more to develop, may continue an infection, and may require operative or cystoscopic treatment. The careful obstetrician can minimize these after-effects to a great extent, and so prevent the development of a serious threat to the health of many women.

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ANTIBODIES TO THE POLIOMYELITIS VIRUSES AT DIFFERENT AGES IN THE MELBOURNE POPULATION.

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SINCE 1951 the tissue culture methods introduced by Enders, Weller and Robbins (Enders *et alii*, 1949; Weller *et alii*, 1949; Robbins *et alii*, 1950) have been used at these laboratories for the isolation of poliomyelitis virus strains and for the detection of serum antibodies to the three known virus types. The findings in patients in Melbourne suffering from poliomyelitis were recently reported (Bazeley and Thayer, 1954*a*; Thayer and Ferris, 1954). This paper is concerned with antibodies present, in relation to age, in the population of Melbourne. It reports the results of the examination of 483 sera from persons of various ages, none of whom had suffered, as far as was ascertainable, a clinical attack of poliomyelitis.

Material and Methods.

Collection of Serum.

The opportunity to carry out this survey was facilitated by the availability of a large number of cold-stored (4° C.), preservative-free specimens of serum which had been collected during investigations by various workers in these and other laboratories.

Serum had been collected at random from persons of various ages, representing a wide cross-section of the population of Melbourne, over the four-year period from September, 1950, to August, 1954; during this time, as was shown by virus isolations, all three virus types were responsible for clinical cases of poliomyelitis in Melbourne.

Thirty-five specimens of serum from babies aged six to eighteen months, consisted of the following: 20 specimens obtained during a pertussis vaccine trial in 1950, eight collected at the Walter and Eliza Hall Institute in 1950, and seven collected at the Royal Children's Hospital in 1953.

Two hundred and sixty-two specimens from children aged from two to sixteen years came from the following sources: 24 collected at the Walter and Eliza Hall Institute in 1950; 93 obtained during a diphtheria vaccine trial in 1951; 76 routine serum samples from children admitted to the Royal Children's Hospital from June, 1952, to June, 1953; and 69 from a combined diphtheria-tetanus vaccine trial in 1954.

Forty-seven specimens from persons aged from sixteen to twenty years were collected during 1954 from 32 trainee nurses and 15 footballers in order to amplify the numbers in this age group.

Ninety-three specimens from persons aged from twenty to fifty-nine years were collected from out-patients and nurses at the Heidelberg Repatriation Hospital and from nurses at the Alfred Hospital during a tuberculosis survey in 1950.

The remaining 46 sera were from random samples collected by this laboratory or from non-poliomyelitis patients admitted to the Fairfield Hospital during the period 1952 to 1954, the ages ranging from one to fifty-nine years.

Poliomyelitis Virus Type Strains.

As in previous reported work (Bazeley and Thayer, 1954*b*), the following poliomyelitis virus type strains were used: Mahoney, Type I (Brunhilde), MEF 1, Type II (Lansing), and Saukett, Type III (Leon). These consistently produce cytopathogenic changes in suitable tissue cultures.

Detection of Antibodies to Poliomyelitis Virus Types.

All sera were tested at a dilution of 1:4; this figure expressed the dilution of serum that was added to the virus before inoculation into the culture tube, such dilution being made in inactivated horse serum (heated at 56° C. for thirty minutes). Evidence of the presence of a poliomyelitis type antibody was determined by the neutralization of the cytopathogenic effect produced by a specific poliomyelitis virus type in tissue culture. A negative result does not prove the absence of antibody or eliminate the possibility of previous exposure to the virus, but merely shows that less than a certain selected level of antibody was present.

The greater portion of this survey was carried out in rhesus monkey testicular tissue cultures. Small portions of testicular tissue were embedded in fowl plasma clots and immersed in a nutrient medium consisting of chick embryo extract, horse serum and Hanks's balanced salt solution (Bazeley and Thayer, 1954*b*). For the remainder of the survey trypsinized rhesus monkey kidney tissue cultures (Bazeley, 1953, personal communication) and the synthetic medium number 199 (Morgan, Morton and Parker, 1950) were used.

After the removal of the medium, to each of the duplicate culture bottles showing satisfactory outgrowth of cells was added an inoculum of 0·1 millilitre prepared by adding 0·15 millilitre of the poliomyelitis type virus suspension, with a virus strength of 100 tissue culture infective doses, to an equal volume of the 1:4 serum dilution. After being shaken, the serum dilution-virus suspension mixture was allowed to stand at room temperature for one hour before use. Ten minutes after inoculation of the tissue culture one millilitre of fresh medium was added and the culture reincubated at 36° C.

Four or six days after inoculation, for kidney or testicular tissue cultures respectively, the cultures were examined under 50-fold magnification, and all showing typical signs of virus-induced degeneration were considered infected. The non-neutralization of the cytopathogenic effect was considered an indication of lack of antibody in the serum to the specific poliomyelitis type virus used.

The tests included uninoculated tissue culture controls and virus titrations to ensure a virus strength of 100 tissue culture infective doses for each poliomyelitis virus type at the time when the test result was read.

During the survey 93 sera were retested and results were consistent with those originally determined, so it is thought that a satisfactory standard was maintained.

Results.

Table I gives the complete results obtained, and shows, for the various age groups, the percentage incidence of single antibodies and multiple antibody combinations to the three poliomyelitis virus types.

Until the age of twenty years the grouping is in five-years periods; beyond this age, in ten-year periods because of the smaller number of serum samples. With increasing age the percentage decline in persons without antibodies and the rise in persons exhibiting multiple antibodies is clearly shown. In view of these changes it is of interest to note that, except for the group aged fifty to fifty-nine years, there is a relatively constant percentage of persons with a single antibody.

The consistently higher percentage with antibody to Type II or III virus relative to Type I in those persons possessing a single antibody, is reflected in those persons possessing two antibodies. The percentage of persons with antibodies to both Types II and III is consistently higher

TABLE I.

Distribution of Antibody to Poliomyelitis Virus (Types I, II and III) in 483 Persons, Melbourne, 1950 to 1954.

Age. (Years.)	Number of Samples.	Specimens Containing Antibody to One Type Only.			Specimens Containing Antibody to Two Types.			Specimens Containing Antibody to All Three Types.	Specimens Containing No Antibody.
		Type I.	Type II.	Type III.	Types I and II.	Types I and III.	Types II and III.		
0 to 4 ..	92	3%	11%	8%	1%	1%	5%	3%	68%
5 to 9 ..	98	7%	13%	6%	4%	3%	9%	5%	53%
10 to 14 ..	104	7%	11%	8%	4%	5%	12%	4%	49%
15 to 19 ..	76	5%	10%	4%	4%	10%	14%	14%	39%
20 to 29 ..	44	14%	7%	7%	9%	9%	11%	27%	16%
30 to 39 ..	23	0%	4%	22%	13%	9%	18%	30%	4%
40 to 49 ..	22	5%	9%	5%	18%	9%	9%	41%	4%
50 to 59 ..	24	0%	0%	4%	4%	17%	25%	46%	4%
Total ..	483	6%	10%	7%	5%	6%	11%	12%	43%

than the percentage of those with the Types I and II or Types I and III combination.

The percentages of positive results to tests for antibody to each virus type at various ages is shown in Table II.

The percentage of persons possessing Type I antibody is consistently less than the percentage of those with Type II or Type III antibody. Of the 483 persons examined, 142 possessed antibody to Type I, 186 to Type II, and 177 to Type III. The deficiency of positive results for Type I antibody relative to Type II or Type III is significant in each case.[1] The difference in incidence of antibodies to Types II and III is not significant.

Interpretation of Findings.

It is conceivable that this lower incidence of Type I antibody may be related to the conditions of the test. If, for example, Type I virus was more cytopathogenic in tissue cultures than the Type II or III strains, then relatively more antibody might be required to neutralize its activity. In this case the deficiency should be the same irrespective of the age of the subjects from whom the samples of serum were obtained.

TABLE II.

Distribution of Antibody to Poliomyelitis Virus (Types I, II and III) in 483 Persons, Melbourne, 1950 to 1954.

Age (Years).	Number of Samples.	Percentage of Samples Containing Antibody.		
		Type I.	Type II.	Type III.
0 to 4	92	9	20	16
5 to 9	98	19	31	23
10 to 14	104	19	32	29
15 to 19	76	33	41	41
20 to 29	44	59	55	55
30 to 39	23	52	65	78
40 to 49	22	73	77	64
50 to 59	24	67	75	92
Total	483	29	38	36

But in fact, as is shown in Table II, the differences in the percentages of positive findings are most pronounced at the earlier years of life, and diminish with advancing age so that they become insignificant in the older age groups. This phenomenon is what would be anticipated if the mean annual attack rate of Type I virus was lower than that of Types II and III, because even if the attack rates of two agents differ, eventually both will succeed in infecting practically all those exposed to infection.

Turner *et alii* (1950) have provided means for calculating mean annual attack rates or, more properly speaking, conversion rates from data such as are here presented. They examined the serum of 970 persons for antibody to Type II (Lansing) virus, using the mouse as the test animal for virus neutralization tests. They considered the rates at which positive findings in infants associated with maternal antibody are lost, and the rates at which positive findings accrue in response to infection and decline in the absence of reinfection. From their data they concluded that positive findings for Type II antibody were lost at the rate of 2·15% *per annum*.

Curves constructed according to their equations showed reasonably good fit when applied to the data of other workers. By the use of their formula and the acceptance of their constants, the mean annual attack, or conversion, rates for Melbourne are calculated as follows: Type I, 3·9%; Type II, 5·2%; Type III, 4·8%.

The estimated mean annual attack rate of Type II virus in Melbourne is considerably lower than most of the rates quoted by Turner *et alii*, varying in North American samples from 20% to 40% *per annum*. However, the findings of Brown and Francis (1947) for 287 serum samples collected in several North American areas during the period from 1941 to 1945 are similar to those for Melbourne.

[1] Type I versus Type II: $\chi^2 = 8{\cdot}95$; $n = 1$; $P < 0{\cdot}01$.
Type I versus Type III: $\chi^2 = 5{\cdot}75$; $n = 1$; $P < 0{\cdot}02$.

Discussion.

It seems that the mean annual attack rates of all three types of poliomyelitis virus are relatively low in Melbourne. The only proviso relating to this statement is that the serological tests were performed by the tissue culture technique, whereas Turner and other workers quoted in his paper used the mouse protection method. However, there is no reason to suppose that one technique is less sensitive than the other. Li and Schaeffer (1954) have noted a close correlation between the results of mouse protection and of tissue culture neutralization tests.

Although the calculated mean annual attack rates are low, the incidence of paralytic poliomyelitis in Melbourne is high. In recent years there have been several large epidemics; the epidemic of 1949 was caused by Type I virus; the 1952 epidemic was caused by Type II virus, and since then Type I strains have been isolated from the majority of patients examined (Thayer and Ferris, 1954).

Low annual attack rates are, of course, consistent with high epidemicity and with a shift in incidence of infection towards the older age groups, as has been noted in Victoria in recent years.

Summary.

Four hundred and eighty-three specimens of serum, collected over a period of four years from random selected Melbourne persons aged from six months to fifty-nine years, were tested in rhesus monkey tissue cultures for neutralizing antibodies to the three poliomyelitis virus types.

A significant difference in positive findings for Type I antibody (29%) in relation to Type II (38%) or Type III (36%) was found. No significant difference occurred in the incidence of antibodies to Type II or Type III poliomyelitis virus.

The mean annual attack or conversion rates for Melbourne are calculated to be as follows: Type I, 3·9%; Type II, 5·2%; Type III, 4·8%.

Acknowledgements.

The writer is indebted to Dr. S. Fisher and Dr. S. Wiener, of these laboratories, to Dr. A. A. Ferris and Dr. W. J Stevenson, of the Fairfield Epidemiological Research Unit, to Dr. Gray Anderson, of the Walter and Eliza Hall Institute of Medical Research, and to Dr. J. Perry, of the Royal Children's Hospital, for their cooperation in supplying specimens of serum. The valued advice of Dr. E. V. Keogh in the preparation of this manuscript is gratefully acknowledged.

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REPARATIVE SURGERY OF THE EXTERNAL NOSE.

By ERIC GUTTERIDGE,
Melbourne.

THE external nose is shaped as that architectural structure termed the hood. This feature projects from a sloping roof in a forward and downward declination, with high-pitched sloping roofs meeting in a gable, and terminating in a gable end or window.

The external nose projects from a recessive fronto-maxillary curve, the face; it has acutely inclined walls ascending to the gable of the bridge of the nose and terminates in twin windows, the nostrils. Structurally, the external nose has the characteristic build of a king post truss, employed to roof large spans requiring strength and stability (Figure I). The rafters supporting the roof coverings meet at the ridge rafter, in a gable formation. The ridge rafter is upheld by a central king post, borne on a transverse tie beam, connecting the bases of the roof walls.

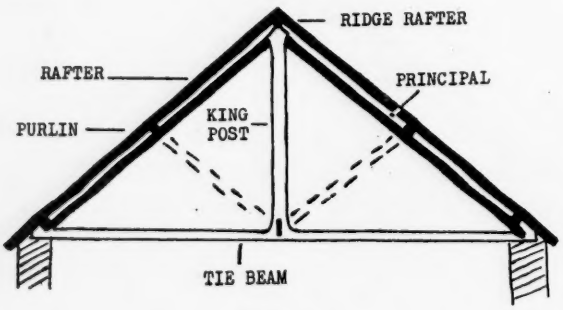

FIGURE I.
King post truss.

The bridge of the nose represents the ridge rafter, the nasal septum matches a series of king posts in parallel, and sustains and braces the nasal bridge. The nasal septum abuts on and is sustained by the nasal floor of the palatal processes of the maxilla and palate bones, corresponding to the horizontal tie beam of the truss.

The truss has roof rafters, stretching from tie beam to ridge rafter, which are laid upon purlins, borne on principals, inner rafters extending from base plates to ridge rafter. This web of timber is mirrored in the bones and cartilages which form the supports of the nasal side walls—the nasal bones and the frontal processes of the maxillæ cranially, the upper and lower lateral cartilages in the middle and lower thirds respectively. The upper two-fifths are of bone and rigid; the lower three-fifths are plates of cartilage and pliant.

The roof covering of the external nose is formed of the integuments, the skin firmly bonded to the periosteum of the nasal bones and the perichondrium of the lateral and orificial cartilages.

Deformities of the nose are distortions of the architectonic components of the external nasal structure, deviations from the pattern of the perfectly shaped nose. The nasal bones as partners with the bony bridge form the bony

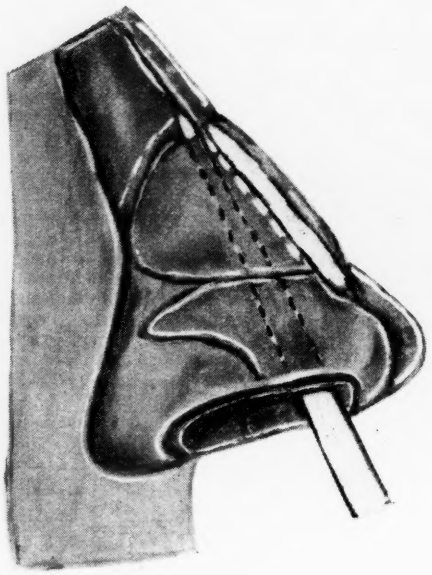

FIGURE II.
Incision at lower border of nasal bone.

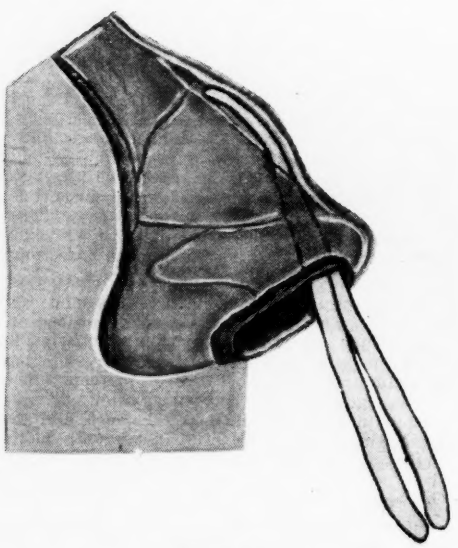

FIGURE III.
Removal of nasal hump with rongeurs.

vault of the nose. Displacements are lateral, with bossing projection of the nasal bones on one or both sides, or concavity associated with convexity of the obverse side. The bridge of the vault may be bossy from injury or inheritance, concave in the dished face, its margins broad and knobby. The middle two-fifths of the bridge is the anterior border of the cartilaginous septum and may be deviated from the centre line, prominent or concave. The

total length of the nose may be excessive (the long nose). The nostrils may be over-wide or narrowed, the nasal tip prominent. Deviations of the nasal septum are distortions of the important king post support of the nasal bridge,

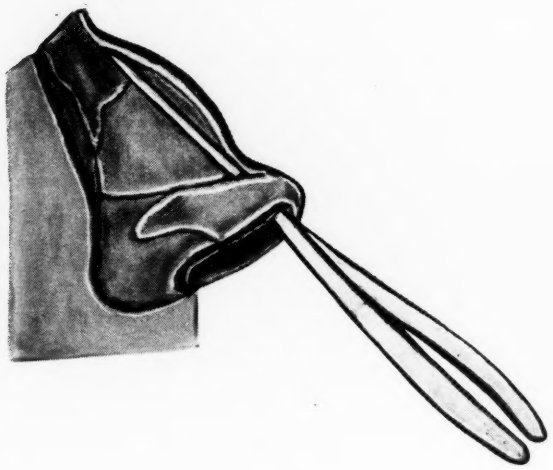

FIGURE IV.
Removal of nasal hump with bone-cutting forceps.

and are commonly associated with, and the result of, traumatic deformities of the bridge.

Reparative surgery of minor deviations and deformities of the external nose should accompany nasal septal

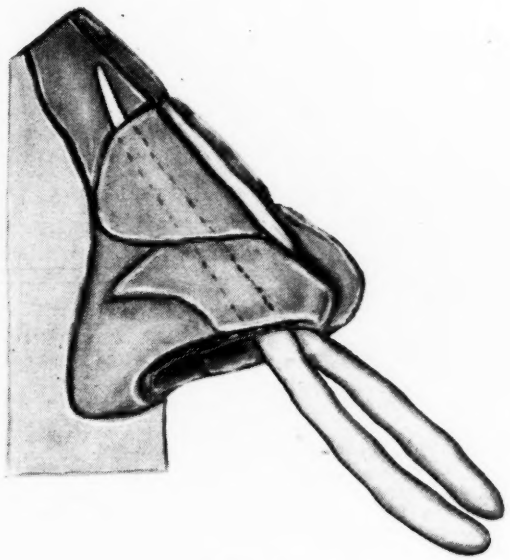

FIGURE V.
In-fracturing of nasal bone.

operations. A limited operative procedure is described to correct such abnormalities and distortions of the nasal external structure.

The Incisions.

The incisions are shown in Figure II.

In contrast to the classical Joseph line of incision (at the lower or caudal border of the upper lateral cartilage) the cartilages are left intact and the incision is made at the caudal border of the nasal bone. The knife passes

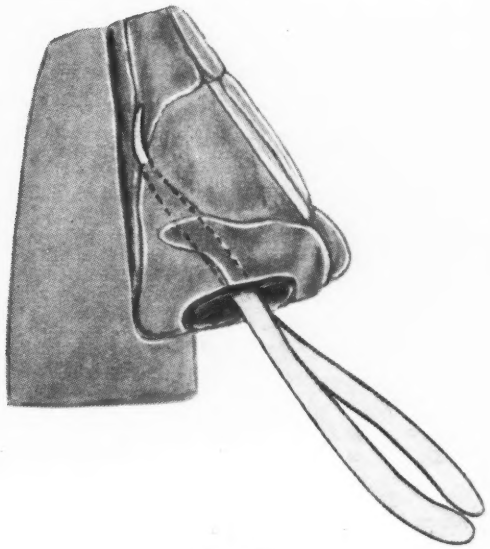

FIGURE VI.
Division of nasal bone.

beneath the skin over the nasal bone to the limit of the bony excrescence or deformity. The undermining may be continued over the nasal process of the superior maxilla

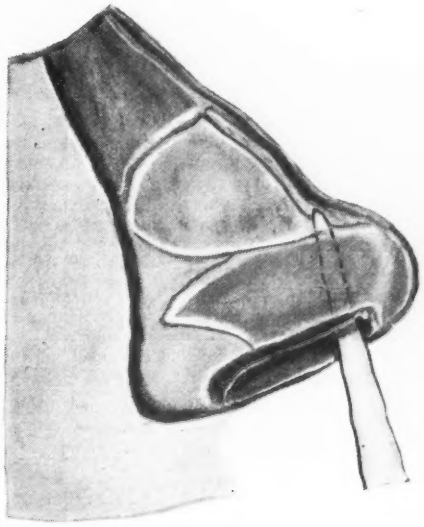

FIGURE VII.
Elevation of subcutaneous tissue for cartilage inlay.

if this is protuberant. The knife is then swept over the *dorsum of the nose over the bony and cartilaginous bridge,* as far as the tuberosity or hump of the nose extends, the skin being raised from the bridge. A similar incision

with elevation of the skin is performed on the opposite side of the nose and the incisions are joined.

The attachment of the lateral cartilages to the septum will be divided by the incision along the cartilaginous bridge, in the cranial portion. This provides a line of drainage, diminishing post-operative swelling. The periosteum over the nasal bones and nasal processes of the maxillæ is not elevated, so that the nutrition of the bones is maintained and the bleeding diminished.

Removal of the Hump or Protuberance.

The method of removal of the hump or protuberance is shown in Figure III. A small bone-cutting *rongeur* is

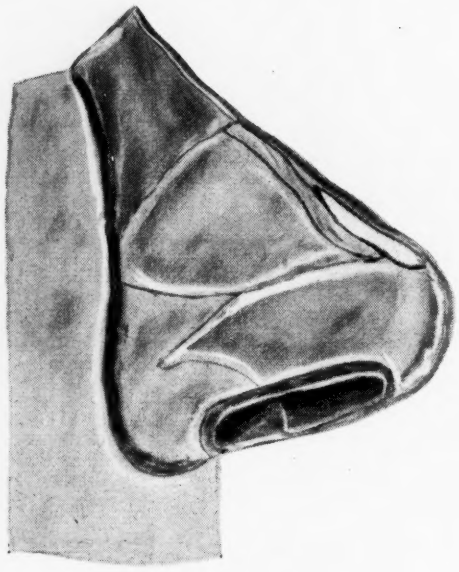

FIGURE VIII.
Cartilage inlay.

introduced through one of the nostrils into the subcutaneous space superficial to the humped bridge, and the jaws are closed upon the lower part of the hump, so that it is wrenched free of periosteal fibres. Further bites are taken until the bridge is cut to the desired straight profile. A narrow bone-cutting forceps may be preferred to grasp the whole length of the hump and remove it in one piece (Figure IV). The bony bridge may be narrowed by shaving of the margins with the *rongeurs*. The cartilaginous bridge may require paring of the dorsum or edges with *rongeur* or knife. A smooth rolled border is desirable.

Inplacement of the Lateral Walls of the Bony Vault.

Removal of the hump leaves a flattened nasal bridge. The upstanding medial borders of the nasal bones require inplacement (Figure V). A *rongeur* grasps the border of the nasal bone outstanding, with one blade through the incision and thus beneath the skin, and the second blade over the mucous membrane in the nostril. The bone is fractured inwards, and remains *in situ*. Further infracture is performed until the nasal bones show a smooth slope from the naso-labial fold to the bridge.

If there is a gross deformity of the vault, involving the nasal process of the maxilla, the bone will require division along the line of the naso-labial fold. With a small-bladed knife the mucous membrane is separated from the bone along the projected line of fracture, and the space is deepened with a small, narrow elevator to form a tunnel between the bone and muco-periosteum (Figure VI). A curved bone-cutting forceps is inserted with one blade in this tunnel and the second external to the bone in the subcutaneous pocket. The nasal process is divided or crushed in two or three bites, and the separation completed with strong short-bladed scissors. A chisel may be required for thicker bone. The process is then grasped with *rongeurs* and infractured to the desired situation.

Inplacement of the Upper Lateral Cartilage.

The attachment of the lateral cartilages to the nasal septum may require division to restore the symmetry of the cartilaginous vault. A continuation of the incision

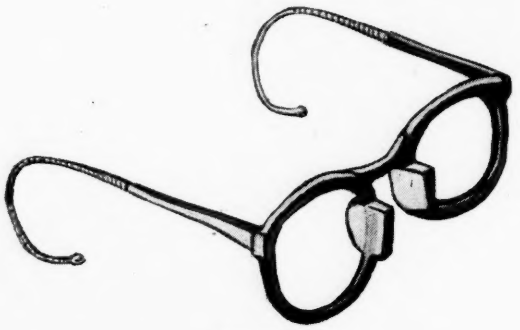

FIGURE IX.
Spectacle splints (made by Optical Prescriptions, Melbourne).

along the dorsum of the nose to the anterior border of the lateral cartilage will separate the cartilage from the nasal bridge and permit the cartilage to move in toward the centre line, obliterating the bulge outward. The knife is in the nasal cavity in this incision and will divide mucous membrane and cartilage from the nasal cavity outward. As there has not been any detachment of the skin and perichondrium overlying the lateral cartilage, the nutrition of the cartilage will not be impaired.

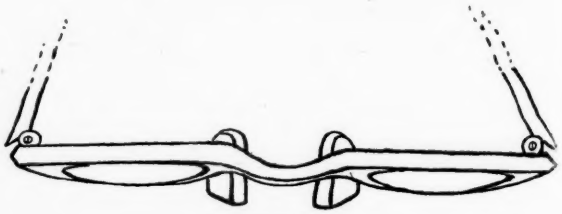

FIGURE X.
Spectacle nasal splints.

Shortening of the Nose.

The original incisions over the nasal bones and bridge are extended over the length of the septum to the anterior or caudal border, the two incisions are joined, and the knife is brought down along the caudal border of the septum to the nasal spine, the columella being thus separated from the septum. A knife or pair of scissors removes the excessive portion of the overlong septal cartilage. Two mattress sutures of tantalum wire ("Ethicon", 0·005 inch diameter) pass from one to the other nostril through septal cartilage and columella. Tantalum is non-irritant and may remain indefinitely.

The tip of the nose, the skin and subcutaneous tissue, firmly attached to the lower lateral cartilages, rises above the bridge line, and a small cartilage inlay may be required to restore the contour. The cartilage is inserted through the incision over the bridge and lies cranial to the lower lateral cartilage.

Cartilage Inlay in the Nasal Bridge.

The portion of the nasal bridge between the nasal bones and the lower lateral cartilages—the penultimate fourth of the bridge—is maintained by the projection of the nasal septal cartilage. Depression of the contour line will be obliterated and the plane of the bridge restored with an inlay of cartilage from a nasal septal cartilage. If a formal septal resection has been performed, the spokeshave will have provided sufficient cartilage. If this has not been done, a small resection will furnish an adequate supply. Heteroplastic preserved cartilage in acriflavine (1 in 1000 emulsion) from the refrigerator is convenient.

A strip of cartilage is shaped, of the width of the bridge and of a length to fill the depression. The ends are bevelled to an acute angle to avert formation of a ridge. The cartilage is immersed in acriflavine emulsion as an antiseptic precaution. A scalpel is inserted from the nasal cavity at the caudal border of the upper lateral cartilage at the point of its attachment to the bridge (Figure VII), passes beneath the skin over the bridge and makes a pocket to receive the cartilage graft. Forceps place the graft through the incision (Figure VIII).

Dressing.

A glove finger filled with gauze is inserted into each nostril and firmly packed. Strips of "Elastoplast" are laid across the nasal bridge. Two small rolls of "Elastoplast" are placed over the sides of the nose and secured in place with a long strip of "Elastoplast" across the nose to the malar eminences.

The intranasal glove fingers are removed after twenty-four hours. The "Elastoplast" strips are adjusted and tightened, and retained for four days.

Penicillin is injected daily.

A minimal amount of reactionary swelling appears over the bridge from subcutaneous œdema and hæmorrhage; this is controlled by the continuous pressure of the "Elastoplast". The small subcutaneous pockets superficial to the nasal bones and frontal processes of the maxilla drain through the intranasal incisions at their free caudal borders. The periosteum over these bones has been preserved intact, and therefore there is little hæmorrhagic exudation. The upper lateral cartilages have been incised at their junction with the nasal septum, but are otherwise intact and undisturbed.

On the fifth day the "Elastoplast" outer strip and side rolls are removed. Over the "Elastoplast" covering of the nose a spectacle splint is worn for four hours daily (Figures IX and X). The spectacle frame has the nasal shoulders or plackets built forwards and downwards to form a rectangular splint to press upon the sides of the nose through the "Elastoplast" covering. The spring arms and the weight of the spectacles and plain lenses provide a constant moderate pressure.

On the sixth day the "Elastoplast" is removed. The spectacle splint is worn for two further days.

DETERMINATION OF MERCURY IN URINE, WITH RESULTS IN CASES OF PINK DISEASE.

By F. R. BARRETT,

School of Public Health and Tropical Medicine, University of Sydney, Sydney.

A METHOD was required for the estimation of mercury in infants' urine. In general, the methods described in the literature for the determination of mercury in biological materials (for example, Laug and Nelson, 1942; Hubbard, 1940; Milton and Hoskins, 1947; Simonsen, 1953) depend upon a digestion of organic matter in the sample, extraction with diphenyl-thiocarbazone (dithizone) or di-β-naphthyl-thiocarbazone and measurement of the coloured mercury complex formed.

It is generally considered that some copper is co-extracted, and various procedures have been used to overcome such interference. Maren (1943), on the other hand, states that "copper need not interfere if the proper conditions of pH are maintained".

In one of their studies with dithizone, Irving and his co-workers (1949) have given pH-extractability curves for mercury and copper and have examined the influence of halide ions and time of shaking upon the extractability of these metals. They could not confirm Marens' statement that "in 50 c.c. [of] 0·25N-hydrochloric acid, 1000 mg. of copper does not change the colour of dithizone reagent", and state that "mercury is seriously over-estimated when an equal or greater weight of copper is present".

The relative positions of the mercury and copper curves along the pH axis, for solutions in chloroform, show little or no overlap, the curve for mercury lying in a more acid region than that for copper. Further, various workers, particularly Barnes (1947), have shown that copper extraction equilibrium is only slowly attained with dilute chloroform solutions. On the other hand, the extractability curves have a sigmoid character, and Irving *et alii* (1949) have also shown that a variable, in contrast to a constant, excess of reagent leads to a distorted form of extraction curve which will lie at a lower pH.

Copper is present in urine in trace amounts only (less than two microgrammes in the urine digests used by the author). From the analytical standpoint, the available evidence does not reveal whether such small amounts of copper would interfere with determinations of varying amounts of mercury.

Experiments were undertaken on dilute standard solutions and on aliquots of digests representing 20 millilitres of urine, to determine whether, under certain conditions of pH, of dithizone concentration in chloroform, and of shaking time, copper is co-extracted when present in trace amounts only, particularly in the presence of small quantities of mercury.

The influence of other substances which have been reported to interfere—namely, chloride ions, manganese (from permanganate digest reagent) and hydroxylamine (reducing agent)—was also examined. For example, compensating errors may have been introduced under the conditions of extraction employed by Maren (1943)—namely, 0·25N hydrochloric acid and short shaking time (50 shakes).

Digestion procedures were modified in accordance with the data obtained from the experiments to be described and in order to maintain hydrogen-ion and salt concentration of digests as uniform as practicable.

Experimental Investigations.

Extractability of Copper.

The following solutions were used: (i) 100 millilitres of 1N sulphuric acid containing five microgrammes of mercury, one millilitre of 50% hydroxylamine hydrochloride and varying amounts of copper; (ii) 100 millilitres of urine digests, 0·2N in chloride ion, containing five microgrammes of mercury after subtraction of the reagent blank, and varying amounts of copper.

The solutions were shaken vigorously for one minute (200 shakes) with 10 millilitres of dithizone solution in chloroform (five milligrammes per litre). The pH of the aqueous phase was approximately 0·7.

The results are recorded to the nearest tenth of a microgramme (Table I).

The error due to copper was then tested with 100 millilitres of solutions containing (*a*) 50, (*b*) 20 or (*c*) 10 microgrammes of copper and varying amounts of mercury. The results are shown in Table II.

Similar results were obtained with urine digests. Reagent blanks, containing 0·3 microgramme of mercury per 100 millilitres, and (i) 10 and (ii) 20 microgrammes of added copper, gave copper errors, expressed as mercury, of 0·1 and 0·2 microgramme respectively.

Effect of Chloride Ions in Reducing Extractability of Mercury.

One hundred millilitres of solutions of normal sulphuric acid containing five microgrammes of mercury, one millilitre of 50% hydroxylamine hydrochloride and varying amounts of sodium chloride, were shaken for (i) forty-five seconds or (ii) sixty seconds with 10 millilitres of dithizone in chloroform (five milligrammes per litre). The results are shown in Table III.

TABLE I.

Mercury Found. (Microgrammes.)	Copper Taken (Microgrammes).		
	0	100	1000
First solution ..	5·0 5·0	5·1 5·1	5·6 5·8
Second solution ..	5·0 5·1	5·1 5·2	5·7 5·9

Effect of Manganese.

The usual dilute standard solutions were prepared, containing five microgrammes of mercury, with the addition of varying amounts of purified manganous sulphate, the volume being maintained constant. The results are shown in Table IV.

TABLE II.

Mercury Level. (Microgrammes.)	Copper Error (Returned as Mercury). (Microgrammes.)		
	Solution (*a*).	Solution (*b*).	Solution (*c*).
5·0	0·0 0·0	—	—
2·0	0·1 0·2	—	—
1·0	0·2 0·2	0·1 0·1	—
0·5	0·2 0·3	0·2 0·1	0·1 0·0

Effect of Excess Hydroxylamine.

Dilute standard solutions containing five microgrammes of mercury and varying amounts of 50% hydroxylamine hydrochloride were shaken for sixty seconds. The results are shown in Table V.

TABLE III.

Mercury Extracted.	Normality of Sodium Chloride Solution.						
	0	0·1	0·2	0·25	0·3	0·35	0·4
Method—							
(i) ..	100%	100%	98%	97%	96%	94%	90%
(ii) ..	100%	100%	100%	99%	98%	96%	94%

Method.

All reagents should be of recognized analytical purity.

Metal-free water is needed. Laboratory distilled water must be redistilled from an all-glass "Pyrex" still.

Diluted sulphuric acid means five hundred millilitres of sulphuric acid of a specific gravity of 1·84 per litre.

For the sulphuric acid–nitric acid mixture, equal volumes of the concentrated acids are used.

Potassium permanganate is provided by "Analoid" tablets (0·5 gramme).

The hydroxylamine hydrochloride used is a 50% weight for volume solution, metal-free; it is purified according to the method of Sandell (1950), the appropriate quantities being used.

To prepare the chloroform, redistil British Pharmacopœia grade from an all-glass "Pyrex" still, and add 1% absolute alcohol which has been distilled over potassium hydroxide.

The dithizone is prepared by purifying good quality commercial reagent according to the method of Sandell (1950). The dithizone solutions are as follows. Stock solution consists of five milligrammes in 100 millilitres of freshly distilled chloroform. It is stored in a dark bottle in the refrigerator and prepared fortnightly.

For the preparation of the extraction solution, stock solution is accurately diluted 1:10 with purified chloroform.

Standard mercury solution, 0·100%, is prepared in accordance with the method of Sandell (1950). Dilute standards are prepared by diluting it with normal sulphuric acid after the addition of one millilitre of 50% hydroxylamine hydrochloride solution per 100 millilitres of solution.

TABLE IV.

Mercury Extracted.	Manganous Sulphate Solution.				
	2·5%	3%	6%	9%	12%
Method (i) ..	100%	99%	97%	95%	94%
Method (ii) ..	100%	100%	98%	96%	95%

Apparatus.

"Pyrex" glassware should be used.

Boiling flasks of 500 millilitres' capacity, with a neck nine inches long and an internal diameter of one and a half inches, are required, with close-fitting, all-glass, water-cooled, drop-in condensers reaching into the body of flasks.

Centrifuge tubes of 50 millilitres' capacity, to which are fitted small condensers, are employed, glass joints being used.

Rinse glassware well with nitric acid and metal-free water before use. Pipettes can be conveniently washed in the glass automatic pipette washer of Barrett and Golding (1951) and finally rinsed with metal-free water.

Corks and rubber stoppers must not be used. The tops of reagent bottles and other containers should be covered to exclude dust.

TABLE V.

Mercury.	50% Hydroxylamine Hydrochloride Solution. (Millilitres.)			
	1	2	3	4
Amount extracted	100%	99%	97%	95%

A suitable photoelectric absorptiometer—for example, the Spekker instrument—is needed. Advice on the use of the Spekker instrument for mercury determinations is given by Barnes (1946). The E.E.L. photoelectric colorimeter, when used with a large capacity two-volt battery and Ilford bright spectrum series filter number 623 (centre 490mμ), is satisfactory.

Procedure.

Digestion.

Measure 40 millilitres of urine into a 500-millilitre boiling flask fitted with "cold-finger" condenser. (If less urine is taken, dilute to 40 millilitres.) Add 12 millilitres of 1:1 sulphuric acid and one tablet (0·5 gramme) of potassium permanganate. Replace the condenser and boil the mixture gently. When the solution clears, quickly add another tablet. Repeat the process until the permanganate colour persists and any fatty material has been completely oxidized. (Towards the end of digestion it is advisable to wash down with a little water before lifting the condenser to add further permanganate tablets.) When digestion is complete, cool the solution, decolorize it slowly with 50% hydroxylamine

hydrochloride solution and add two millilitres excess. Replace the condenser and boil the solution gently for one minute. Cool it under running tap water and dilute to 200 millilitres.

If more than ten tablets of permanganate are required, dilute the solution accordingly, after adding the requisite amount of 1:1 sulphuric acid and excess hydroxylamine solution.

Run a reagent blank, using 40 millilitres of water in lieu of a sample.

For urine specimens containing much protein, proceed as follows:

Measure 40 millilitres of urine into a centrifuge tube fitted with a small condenser. Adjust the pH to about 5·4, the isoelectric point of denatured albumin (*vide* Cole, 1933). Place in a water bath and boil under reflux until the proteins coagulate. Cool the mixture and readjust the pH to about 5·4 if necessary. Centrifuge at 4000 revolutions per minute for fifteen minutes. Decant and drain the supernatant fluid into another container for later digestion.

Measure eight millilitres of sulphuric-nitric acid mixture into a small vessel. Add about two millilitres of the mixture under the precipitate with a Pasteur pipette. The precipitate will float on the heavier fluid. Decant quickly *en masse* into a dry boiling flask fitted with a drop-in condenser. With the Pasteur pipette, rinse the centrifuge tube with small portions of the remainder of the acid mixture and add the rinsings to the flask. Add one or two small pieces of "Pyrex" glass for smooth boiling.

Heat gently at first, then raise the heat gradually to avoid spluttering. Dense brown fumes of oxides of nitrogen are evolved. Boil for seventy-five minutes, cool, and add one tablet of potassium permanganate and heat to boiling. Permanganate is reduced by nitrous acid and nitric acid fumes are evolved. Cool.

Add the supernatant. Rinse the container with four millilitres of 1:1 sulphuric acid and add the rinsings to the digestion flask. Add permanganate, a tablet at a time, and continue permanganate digestion as described. However, in preparing the digest solution, the excess hydroxylamine should be added cold and just prior to extraction.

Extraction.

Proceed with extraction and measurement without undue delay and in subdued light.

Transfer 100 millilitres of digest solution to a 250-millilitre (bulb) separating funnel. Add 10 millilitres of dithizone solution (five milligrammes per litre) and shake the mixture for at least one minute (200 shakes). If green dithizone colour predominates, proceed under measurement. In all other cases take an aliquot of solution containing approximately five microgrammes of mercury. (A preliminary titration may be necessary.) Dilute to 100 millilitres with normal sulphuric acid, after adding the requisite amount of hydroxylamine solution, and extract as described above.

Measurement.

Run the chloroform extract through a small plug of absorbent cotton wool in the shortened stem of the separating funnel. Collect the clear extract into an eight-millilitre cell and measure at 490mμ by the mixed colour method (that is, in the presence of excess dithizone).

If the chloroform extract is turbid, warm it for a few seconds in warm water. The turbidity may be due to condensed moisture, but should not occur with the above-described limited shaking and with cells at room temperature. The turbidity should not be due to fatty material, which should have been completely oxidized during digestion.

The best results are obtained when two dilute standards are extracted and measured at the same time, to check the calibration curve. The calibration curve should always be checked whenever a fresh stock solution of dithizone is prepared.

Discussion.

The main reasons for each step in the procedure will now be discussed.

Digestion.

Sulphuric acid–nitric acid mixture was found unsuitable for urine, owing to dilution of the acid mixture by the urine. Hubbard's procedure (1940), with the use of sulphuric acid and potassium permanganate tablets, one at a time, was found satisfactory for most samples of urine. However, the composition of urine varies; for example, it may be "concentrated" or it may have a high protein content. Therefore the amount of permanganate and of hydroxylamine required may vary considerably.

The experimental data show that, under the conditions used, mercury can be completely extracted from solutions 0·2N in chloride ion, *plus* one millilitre excess of hydroxylamine hydrochloride solution, and containing 2·5 grammes of manganous sulphate.

It was found possible by the method described to digest completely 40 millilitres of most specimens of urine, including "concentrated" urine, and to extract half aliquots of the diluted digest, without interference from manganese or chloride. In some cases larger amounts of digestion reagents were required. The digest was therefore diluted, as described, to provide digests as uniform in composition and in pH as practicable. The digest solution was used for the duplicate analysis.

Permanganate was found unsuitable for the digestion of urine samples containing much protein, for which a new procedure has been described. The technique for the digestion of the precipitate is adapted from that of Cholak and Hubbard (1946) for tissues. If it is assumed that all the mercury is present as albuminate, it should not be necessary to boil under reflux during heat coagulation. However, owing to insufficient samples of "nephritic" urine containing mercury, adequate testing has not been possible.

It is essential that any fatty material present be removed, for example, by oxidation, otherwise chloroform-soluble colouring matter may be extracted. Alternatively, the digest solution may be washed with a small quantity of chloroform prior to extraction. If so, the dilute standard solutions must be treated similarly.

Chloride.

The conflicting reports can be explained, in part at least, in terms of shaking times. Another aspect to be considered is that raised by Irving *et alii* (1949), that ions of the type $HgCl_3'$ or $HgCl_4''$ may be involved.

Hydroxlamine.

A slight excess of hydroxylamine hydrochloride is generally added to a digest to maintain reducing conditions, since dithizone is sensitive to oxidizing agents. In agreement with Simonsen (1953) it was found that too great an excess hinders the extraction of mercury, unrelated, to some extent at least, to its chloride content.

It would appear advisable to oxidize the sample completely and so limit the amount of excess hydroxylamine required for the maintenance of reducing conditions.

Copper.

Fischer (1937) states that a "mixed colour" dithizone method would be disturbed by the presence of copper, silver, gold, palladium and platinum. With the exception of copper, these metals are rarely present in biological materials, and in the presence of chloride silver "dithizonate" is not formed.

In contrast to copper, mercury is rapidly extracted with dilute solutions of dithizone in chloroform. As would be expected, more copper is extracted with increased concentration of dithizone. With a concentration of five milligrammes of dithizone per litre of chloroform, it was found that mercury complex formation and partition equilibrium could be effected in a reasonable time—namely, one minute (200 shakes) in the presence of the maximum allowable amounts of chloride and manganese, at pH 0·7.

To conform with these conditions, digest solutions (100 millilitres) representing 20 millilitres of urine were used. The amounts of copper normally present in such solutions are in the order of two microgrammes or less (*vide* Hess, Supplee and Bellis, 1923). Better methods for the determination of copper may reveal still lower figures (for example, Eden and Green, 1940; Hubbard and Spettel, 1953). According to Stokes *et alii* (1955), the copper content of urine in Wilson's disease (hepatolenticular

degeneration), one microgramme per millilitre, is 50 to 100 times the normal amount of copper in urine. Even in these rare cases the maximum copper error, as shown experimentally for 20 microgrammes of copper, would be 0·2 microgramme. It would appear that the normal amount of copper in 20 millilitres of urine is only 0·2 to 0·4 microgramme.

Over 200 specimens of "normal" urine have been tested and have returned the same amount of mercury as the reagent blank. Samples of urine from several industrial workers exposed to copper have revealed no copper error.

The direct extraction method is not recommended for the determination of mercury in tissues, gastric contents and fæces.

Measurement.

The various methods for the measurement of mercury dithizonate are discussed by Sandell (1950), including the mixed colour method (that is, an excess of dithizone in the presence of the mercury complex). The mixed colour method was used on the above-mentioned extracts. Care was taken to ensure that there was sufficient excess dithizone by measuring five microgrammes or less of mercury in 10 millilitres of dithizone solution (five milligrammes per litre of chloroform).

With each set of experiments two dilute standards were prepared to check the calibration curve. I prefer to use this method rather than to rely on predetermined calibration curves. Whether this is always necessary will depend on the stability of the dithizone solution. In any case, it is necessary to prepare a new calibration curve each time a fresh stock solution is prepared, say fortnightly. With the methods of extraction and measurement described above, standardization is simple and takes only a few minutes.

Results.

Analytical results are given in Table VI. The results were obtained by analysing a half-aliquot part of the digest solution.

Results from patients suffering from pink disease (acrodynia) are shown in Table VII. Earlier results, together with controls, were reported by Clements (1953).

TABLE VI.

Recovery of Known Amounts of Mercury Added to Urine (40 Millilitre Samples).

Mercury Added. (Micrograms.)	Mercury Found. (Micrograms.)	*Minus* Blank.	Recovery. (*Per Centum.*)	Mean. (*Per Centum.*)
0·5	(i) 1·1	0·5	100	
	(ii) 1·2	0·6	120	113
	(iii) 1·2	0·6	120	
1·0	(i) 1·4	0·9	90	
	(ii) 1·4	0·9	90	93
	(iii) 1·5	1·0	100	
2·0	(i) 2·7	2·1	105	
	(ii) 2·6	2·0	100	105
	(iii) 2·8	2·2	110	
4·0	(i) 4·6	4·0	100	
	(ii) 4·7	4·1	102·5	102
	(iii) 4·7	4·1	102·5	
10·0	(i) 10·5	10·0	100	
	(ii) 10·3	9·8	98	99
	(iii) 10·5	10·0	100	

The figures shown in Table VII are similar to those obtained by Warkany and Hubbard (1948).

Summary.

1. A simple and accurate direct extraction method for the determination of mercury in urine is described.

2. The digestion procedure is based on Hubbard's sulphuric acid–permanganate reagent and on experimental data of interference from digestion reagents and their products. A technique for the digestion of urine samples of high protein content is described.

3. Experimental studies reveal that, under the conditions used, trace amounts of copper present in the urine digests are not co-extracted, irrespective of the amount of mercury present.

4. The direct-extraction technique is not recommended for the estimation of mercury in tissues, gastric contents *et cetera*.

5. The conflicting reports of interference by chloride ions can be explained, at least in part, in terms of shaking times.

6. Results of estimation of mercury excretion in the urine of patients with pink disease (acrodynia) are reported.

TABLE VII.

Urinary Excretion of Mercury in Pink Disease.

Mercury in Urine. (Micrograms per 100 Millilitres.)	Number of Infants in Each Class.	Proportion in Each Class.
0	4	0·09
1 to 10	10	0·22
11 to 20	14	0·31
21 to 30	8	0·18
31 to 40	3	0·07
41 to 50	4	0·09
Over 50	2	0·04
Total ..	45	1·00

Acknowledgements.

This paper is published with the permission of the Director-General of Health, Canberra. I desire to thank the Director of the Institute of Child Health for making available for perusal the hospital records of patients with pink disease.

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Reports of Cases.

INTERSIGMOID HERNIA: A CASE REPORT.

By K. JOHNS,
Newcastle.

ALTHOUGH the intersigmoid fossa is constantly present in infancy and may persist into adult life, the incidence being variously recorded as 52% to 85% of people, reports of internal strangulation due to persistence of this fossa or recess are not common. Up till 1953 Thompson and Robins could find reports of only 26 cases, in 21 of which the condition presented as an acute obstruction. The following case is of interest because of its relative rarity, and also because of the almost complete lack of pain associated with a six-day small bowel obstruction.

Clinical Record.

Mrs. A., aged fifty years, was examined by me at home on November 1, 1954. Five days before she had been perfectly well, when she experienced abdominal pain, mild in nature, lasting about thirty minutes. She went to the toilet and passed a normal motion, which did not relieve the pain. After she had lain down for a short while the pain passed off, and at no other time in the course of the illness did she have any return of pain. She felt a "little off colour", but was able to eat normal meals that day; however, by evening she had commenced to vomit. She vomited intermittently and had absolute constipation from then on. She had undergone a left mastectomy ten years before for chronic interstitial mastitis.

On examination, the patient was a thin woman in no obvious pain; her temperature was 100° F., her pulse rate was 96 per minute, and her tongue was coated and dry. There was slight distension of the lower part of the abdomen, which was doughy, but not very tender. The liver was not palpable, and no jaundice was detected in the skin or conjunctivæ. Loud borborygmi were audible from beside the bed. The vomitus was dark, but not fæculent. The abdomen was resonant to percussion all over, with no shifting dulness. The provisional diagnosis was made of "bowel obstruction ? congenital band", and the patient was admitted to hospital that day. As there were also a large number of cases of infectious hepatitis in the district at the time, the urine was examined for bile and a flat X-ray film of the abdomen was taken on her admission to hospital. The urine test for bile gave a positive result, and the report on the X-ray film was as follows:

> Dilated small bowel loops are shown with fluid separating the coils. No fluid levels are shown within the bowel in the erect position. The appearances suggest intraabdominal fluid with probable obstruction in the distal small bowel.

At this stage, Dr. W. H. Neild was asked to examine the patient in consultation, as the finding of bile in the urine was misleading.

After further observation of the patient in hospital, during which time she continued to vomit, exploratory laparotomy was decided upon. After preliminary intravenous therapy this was carried out on November 2, 1954. At operation a small-bowel obstruction of the terminal portion of the ileum was found, with a loop of ileum imprisoned in an intersigmoid recess deep enough to admit the terminal joint of the index finger. The bowel was plum-coloured but viable, and was released after dilatation of the fossa opening. The hole was closed with interrupted sutures. Continuous gastric suction was started and the intravenous therapy continued, and after twenty-four hours the bowel sounds had returned. The Wangensteen drainage tube was removed on the third day.

The patient made an uneventful recovery, and when she was last examined, in March, 1955, she was perfectly well.

Comment.

Aird reports that in this condition precise diagnosis by X-ray examination with an opaque meal has been made in some cases; it was not attempted here.

Wangensteen, Thorek and Cope all emphasize the value of localized tenderness in the diagnosis of acute obstruction of the small bowel. However, in this case the site of obstruction was inaccessible to abdominal palpation or to rectal examination.

The delay in making a firm diagnosis and in following this by laparotomy was regretted in retrospect; but the complete lack of pain associated with bile in the urine (still unexplained) provided a "red herring" until it was decided to disregard them.

Acknowledgement.

My thanks are due to Dr. W. H. Neild, who examined this patient in consultation with me.

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Reviews.

Clinical Aspects of the Autonomic Nervous System. By L. A. Gillilan, Ph.D., M.D.; 1955. London: J. and A. Churchill, Limited. 8½" × 6", pp. 328, with 42 illustrations. Price: 45s.

THIS book is an evident attempt to make available within reasonable compass information usually locked away in more formidable volumes. Rather more than half the text is devoted to the anatomy and physiology of the autonomic system, together with some clinical comments; the remainder is concerned with clinical observations, together with anatomical and physiological comments. While some judicious repetition is valuable in teaching, that is unnecessary here when the whole book is available for reference backwards and forwards. More rigid editing and some rearrangement could reduce the bulk of words and produce a more straightforward text. The author begins literally at the top, with the brain, and gradually works down. In the cerebral section he lays great emphasis upon the relation of mind to visceral activity and is obviously a strong believer in physical and mental "types" of individuals—but while he mentions that there may be all intervening grades, he subsequently uses such terms as "vagotonic" and "sympathotonic" with a confidence that is not justified by the facts. He rightly emphasizes the abundance and importance of visceral afferent pathways along both sympathetic and parasympathetic nerves, but one questions the advisability of calling any of these "sensory" when many of the impulses they carry never attain the status of "sensation"; "afferent" is less committal and covers the situation adequately. The bulk of the text follows routine accounts fairly closely, but the effort to include everything possible makes it rather synoptic. Insistence upon the importance of parasympathetic efferents in spinal nerves is a matter for controversy, and here Dr. Gillilan has misquoted at least one author—Kiss—who believed, as do most observers, that the small cells in the spinal ganglia belong to visceral afferent fibres. The author's style is not always clear; for example, on page 47 he says "the renal plexus . . . many times receives the least splanchnic nerve directly" when he means, of course, that in many bodies the least splanchnic nerve goes direct to the renal plexus. Another awkward construction occurs on page 48: "The grey rami frequently lie more medial to the spinal cord than do the white rami"; anything lying medial to the spinal cord would be a miracle—presumably "nearer to" is intended. Yet another *bêtise* is "tearing", by which he means "lacrimating" or "weeping", not "ripping". The illustrations—all line blocks—are often not very illuminating. But even more aggravating is the fact that they are not numbered in sequence. This makes it difficult to find a figure from the text reference, and the situation is only slightly improved by the fact that some figures are repeated several times. Surely, even if the

figure order was changed just before publication, there was not so much hurry that the figures could not have been renumbered. The bibliography occupies 27 pages—out of all proportion to the scope of this book—and it contains some defects. One is the misquotation of Kiss's work already mentioned, another relates to the hypophyseo-portal system where Wislocki is given prominence but no mention is made of Popa and Fielding, who discovered the system. Incidentally, the bibliography contains no reference to any original work by the author himself.

To sum up: this promises to become a useful compilation of information upon the autonomic nervous system, but it needs radical revision before it can be recommended with any confidence.

The Living Brain. By W. Grey Walter, M.A., Sc.D.; 1953. London: Gerald Duckworth and Company, Limited. 9″ × 5½″, pp. 228, with 23 illustrations. Price: 15s.

ADAPTING a title of Bernard Shaw's one might well describe this book of Dr. Walter's as "a very intelligent layman's guide to the new science of the electrophysiology of the brain".

Not only the very intelligent layman, but with him the medical practitioner who has been seeking to keep himself up to date is likely to find in this book things both novel and significant, and calculated to require some very close attention; but the effort will be rewarding to a better understanding of the working of that most remarkable of all instruments—the human brain.

The science of neural electrophysiology is very young. Dr. Walter quotes the dictum made at no distant date by a very eminent neurologist who had said that as far as knowledge went of how the brain functioned, the cranium might almost as well have been stuffed with cotton wool as occupied by the brain. Not so since the advent of this new science; and how recent, for it is only twenty-one years since Adrian, in a laboratory in Cambridge, first demonstrated the alpha rhythm, and only about a decade since the first tentative clinical recording in this country of the electroencephalographic machine was made.

As Walter points out, Pavlov, with all his psychological acumen in establishing the facts about conditioned reflexes, was content to leave as quite a closed book how the brain was functioning as a physiological organ.

Now hundreds of workers in many lands are at work decoding the electrical signals that have come to them from the brain as it works, or conversely, as it rests; not only are there data from the electroencephalographic recordings, but now a new and even more spectacular instrument, the toposcope, has been called into being. There, one almost sees, to quote Sherrington, the brain at work as "an enchanted loom where millions of flashing shuttles weave a dissolving pattern, always a meaningful pattern though never an abiding one".

Much light has been thrown upon the mechanisms of the brain by building electrical machines replete with feed-backs and revolving circuits. Walter has exercised his pleasing wit by giving them names suggestive of biological species, for example, *Machina Docilis, Machina Speculatrix, Machina Sapora*. Their conduct is intriguing. They will come when called; they record and "remember"; they "learn" to avoid the path where they have been badly treated; they behave exactly like animals who have become irritated or frustrated.

Not a few readers introduced to this fascinating subject for the first time may well echo the words of a former Head of a Women's College in Oxford, that it is "the most exciting book I have ever read".

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Cancer: As the Disease of Our Time", by Dr. A. Leroi; 1955. London: New Knowledge Books. 7″ × 5″, pp. 16. Price: 1s. 6d.

The author deals with two sets of causes that lead to cancer. The first comprise the "inner and more hidden causes connected with the course of man's destiny"; the second are "those causes connected with the influences that a man receives from his environment".

"Financing Hospital Care in the United States: Volume II. Prepayment and the Community", edited by Harry Becker; 1955. New York: McGraw-Hill Book Company, Incorporated. 9″ × 6″, pp. 380, with 86 illustrations. Price: $4.50.

"Financing Hospital Care in the United States: Volume III. Financing Hospital Care for Nonwage and Low-Income Groups", edited by Harry Becker; 1955. New York: McGraw-Hill Book Company, Incorporated. 9″ × 6″, pp. 128, with 22 illustrations. Price: $2.50.

Published by the Commission on Financing of Hospital Care of the United States of America.

"1955 Medical Progress: A Review of Medical Advances during 1954", edited by Morris Fishbein, M.D.; 1955. New York: McGraw-Hill Book Company, Incorporated. 9″ × 6″, pp. 358. Price: $5.00.

Twenty subjects are discussed in separate chapters.

"Practical Endocrinology", by Lewis M. Hurxthal, M.D., in cooperation with A. Seymour Parker, M.D., and Hirsh Sulkowitch, M.D.; 1955. New York: Landsberger Medical Books, Incorporated. 8″ × 5½″, pp. 318, with 17 illustrations. Price: $7.00.

This is one of a series entitled "Handbooks for the General Practitioner".

"Pathology", by Peter A. Herbut, M.D.; 1955. Philadelphia: Lea and Febiger. 10″ × 7″, pp. 1228, with 651 illustrations and six colour plates. Price: £8 16s.

Intended for undergraduate and post-graduate students.

"Differential Diagnosis of Internal Diseases: Clinical Analysis and Synthesis of Symptoms and Signs on Pathophysiologic Basis", by Julius Bauer, M.D., F.A.C.P.; Second Edition; 1955. New York and London: Grune and Stratton, Incorporated. 9″ × 6″, pp. 1004, with 66 illustrations. Price: $15.00.

The first edition was published in 1950.

"The Principles and Practice of Surgical Nursing", by D. F. Ellison Nash, F.R.C.S.; 1955. London: Edward Arnold (Publishers), Limited. 8½″ × 5½″, pp. 1016, with 371 illustrations Price: 30s.

"Provided a nurse understands the basic principles of her method and takes care to find out *why* a particular practice or routine procedure is adopted, she will not be criticized."

"Proceedings of the Third Medical Conference om Muscular Dystrophy Associations of America, Incorporated, New York, October 8 and 9, 1954", edited by H. D. Bouman; 1955. Baltimore: Williams and Wilkins, Company. 10″ × 7″, pp. 328, with many illustrations.

There are reports on six symposia.

"Forceps Deliveries", by Edward H. Dennen, M.D., F.A.C.S.; 1955. Philadelphia: F. A. Davis, Company (Publishers). 9½″ × 6½″, pp. 242, with 91 illustrations. Price: 71s. 6d.

One of a series of monographs on obstetrics and gynæcology edited by Claude E. Heaton.

"The Cell, The Human Organism and Cancer", by Dr. A. Leroi; 1955. London: New Knowledge Books. 7″ × 5″, pp. 24. Price: 2s. 6d.

The author holds that the causes of cancer should be sought in the organism and not only in the cells.

"How to Overcome Nervousness", by Michael Rogers; 1955. London: New Knowledge Books. 7″ × 5″, pp. 24. Price: 1s. 6d.

Based on the work and teaching of Rudolph Steiner.

"The Surgical Clinics of North America"; 1955. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. Lahey Clinic Number. 9″ × 6″, pp. 307, with many illustrations. Price: £8 2s. 6d. per year in cloth binding and £6 15s. per year in paper binding.

This number consists mainly of a symposium on surgery of the digestive tract; there are 25 articles in this group covering a particularly wide range of subjects. In addition there is a group of five "additional subjects".

The Medical Journal of Australia

SATURDAY, SEPTEMBER 10, 1955.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

RESEARCH IN GENERAL PRACTICE.

ONE of the results of the inauguration of the College of General Practitioners has been the laying of a new emphasis on the possibility of, and the need for, research in general practice. It is not without significance that Dr. W. N. Pickles was elected first President of the College in England. Most people have realized, and this journal has always taught, that it is the prerogative and the privilege of the general practitioner not only to treat a patient suffering from disease and to try to prevent its occurrence, but also to record his observations in a systematic way. It has been pointed out over and over again that the discovery of a single new fact in clinical medicine may be of the utmost importance in the structure of a new building whose coping stone may be placed in position by some other observer at a later date. It is quite possible that delay in the discovery of a material fact may retard the completion of the building for quite an appreciable time. As most readers of this journal are aware, Regional Faculties of the College of General Practitioners have been established in Australia—in New South Wales and in Queensland. At the annual meeting of the New South Wales body, held in September, 1954, it was announced that a research committee had been established to assist practitioners in research which they wished to carry on alone or to help them to form study groups of practitioners who have mutual interests. It may be recalled that in October last year reference was made in these columns to a statement broadcast by Sir Howard Florey that "nearly every procedure in diagnosis, treatment and prevention of disease now applied in the hospital or home took its origin or owes its development to laboratory work". He said this in order to disabuse his hearers of the commonly held view that advances in medical research during the past half-century or so had been mainly or exclusively due to the efforts of those working in hospitals. We thought that this statement was likely to give a false impression to members of the Australian public. We preferred to lay emphasis on Sir Howard Florey's words "owes its development to" and to accept the implication, which we thought Sir Howard Florey himself would be the first to admit, that work completed in the laboratory does not always originate in the laboratory.

This subject comes up for discussion because of the publication of the first John Matheson Shaw Lecture of the Royal College of Physicians of Edinburgh, delivered in November, 1954, by Dr. James M. Mackintosh. This lecture should put heart into general practitioners who may be a little doubtful of their ability to do anything worth while in the matter of research. At the outset, Mackintosh raises the interesting question of the date of the birth of general practice. He thinks that an arbitrary choice of one hundred years is convenient, especially since that period coincides with the introduction into England of the first *Medical Act*. He thinks that this may not be altogether fair, for he asks whether a Jenner or a Ferriar or a William Cullen should be excluded. He points out that the *Medical Act* of 1838, the purpose of which was "to regulate the qualifications of practitioners in medicine and surgery", contained provision for a register compiled of all practitioners qualified to practise. General practitioners were mentioned, but were not conspicuous, either as individuals or otherwise, on the membership of the Council. It was not until 1911 that substantial reference was made by the legislature to general practice. In the *National Insurance Act* of 1911, a "research penny" was provided—one penny for each insured person was to be devoted to research. Incidentally, research was limited to "those diseases from which insured persons suffer". Research in the sphere of public health was started officially by the middle of the nineteenth century, owing largely to the efforts of Sir John Simon. In 1870 he secured the passage of legislation awarding the Privy Council an annual grant of £2000 for "auxiliary scientific investigations". This was the beginning of what became eventually the Medical Research Committee in 1913, and in 1920 the Medical Research Council. In the *National Health Service Act* of 1946 it is stated that "the Minister may conduct, or assist by grant or otherwise, any person to conduct, research into any matters relating to the causation, prevention, diagnosis or treatment of illness or mental defectiveness". In regard to this clause, the Medical Research Council and hospitals are specifically mentioned, and Mackintosh regards it as noteworthy that the Medical Research Council has set up a committee on general practitioner research. In the White Paper on clinical research in relation to the National Health Service, it is noted that clinical research is to include observations in general practice. The following extract is quoted:

> Clinical research ranges from the making of observations which are incidental to, and inseparable from, good practice to systematic investigations undertaken deliberately and often over long periods, with the object of answering specific questions. The former has always been an activity of good practitioners and, apart from seeing that it receives appropriate support, requires no special organization.

Mackintosh thinks that it is a pity that references to the problems specially suitable for research in general practice are so cautious and obscure. He adds that it has been demonstrated beyond question by the College of General Practitioners that research in the field of general practice does require special organization, including statistical and laboratory assistance and the most careful planning. He thinks that it should be added in fairness to the Advisory Committee, whose statement has just been quoted, that it did not assume that research would be carried out solely by men and women who had entered general practice for the avowed purpose of undertaking specialist investigations; it indicated that in appropriate cases the general practitioner might work in a research unit. This, he explains, is a different matter from research in general practice; he thinks that the committee takes rather a gloomy view of the conflict with private practice. The committee stated that modern research had become so exacting an occupation that it was unlikely that many research workers would choose to spend a substantial part of their time in private practice.

Mackintosh points out that during the past decade there has been a new emphasis on research and that the importance of the family doctor is being increasingly recognized. This is more than likely because so many factors tend towards his elimination. He discusses the way in which the general practitioner can be assisted in conducting field research and become a fully accepted member of the research team. He enumerates the agencies which are ready and willing to help in this important organization. The first agency that he mentions is the Public Health Laboratory Service. Applying this statement to Australian conditions, we can enumerate governmental hospital and private pathological laboratories which are always at hand to aid the practitioner. Any practitioner who wishes to follow up observations made on a patient will find experts in pathology ready and willing to help him. Library assistance is also available to him, and as a rule he will experience no difficulty in discovering what has appeared in the literature on the subject in which he is interested. Pathologists are not content to be like penny-in-the-slot machines, but will help the practitioner if he is striving to discover the significance of his observations. If the practitioner records what he has found, together with any documentary evidence that he may discover relating to it, he is in a position to go a step further, if and when some similar or related event arises. The second agency mentioned by Mackintosh is the medical statistician. Any observer who sets out to record observations and draw conclusions from them will be ill-advised if he does not submit his observations to statistical analysis. It is commonly held that undergraduate students in medicine should be instructed in the elements of statistical analysis. To some persons this kind of study does not come easily, but they will generally be able to obtain the assistance of an expert, and they certainly should do so before they make definite statements about their conclusions. The third agency mentioned by Mackintosh is the College of General Practitioners. He quotes the phrase bequeathed to the College by its Steering Committee: "to encourage, guide and coordinate research into the problems of general practitioners". Annual reports of the College of General Practitioners make it "abundantly clear that general practice is rightly regarded as an end in itself, and not an appendage to any hospital or specialty". It is hoped that in Australia regional councils of the College of General Practitioners will encourage and help their members in this matter. It need hardly be pointed out that any general practitioner can direct his energies towards research—he has not necessarily to be a member of the College. Mackintosh describes two types of research. The first he describes as that of "the cat that walks by himself"; this is the man who likes to plod along on his own track. The second type is group research. Mackintosh thinks that this type is very successful within its scope, because it brings in the type of man who is willing to cooperate in research, but does not possess the imaginative power or the time and energy necessary to promote individual or group investigations. Ten types of investigations suitable for general practitioners are named. These are, in order, hereditary defects and diseases, minor ailments, therapeutics, care of the aged, the stress diseases, prognosis, the early states of disease, mental illness, epidemiology and observations on the normal. It must be clear that the general practitioner has an advantage over other observers in two respects. First of all, he sees more of normal persons than other observers do, and secondly, he has the advantage of seeing the early stages of disease. Certain obstacles to general practitioner research are named. Among these are lack of preparation owing to defects in undergraduate training, a want of liaison with consultants and specialists, the difficulties associated with the securing of statistical control, lack of time, the attitude of the public in that they do not enjoy the idea of being the subjects of investigation, record-keeping, and finally, lack of encouragement. Special emphasis should perhaps be laid on the keeping of records. A practitioner will have an initial tactical advantage if he adopts a uniform system of record-keeping, writing his observations while they are fresh in his mind, adopting the scientific method wherever possible, and being logical in his self-criticism so that he does not make a *propter hoc* of every *post hoc*. To draw general conclusions from the results achieved in a single case is a futile and stupid procedure.

We may conclude this short survey with two quotations: the first is from a Research Newsletter of the College of General Practitioners, and the second is from Mackintosh himself. The College writes that there is "a challenge to those in general practice, who see the beginnings of disease, to make a fuller contribution to its study by investigating more fully the problems they handle". Mackintosh writes: "There is a danger inherent in the work of general practice that the very great variety of subjects for practice and thought in a day's work may lead to a superficial and desultory curiosity rather than an eagerness for study. It is easy to spread a thin layer of butter on the bread of idleness."

Current Comment.

MORPHINE-INDUCED INCREASE IN INTRACRANIAL PRESSURE.

NALORPHINE ("Nalline") is an effective antagonist to morphine, and A. S. Keats and J. C. Mithoefer[1] have investigated its effect in preventing the rise in intracranial pressure which morphine produces, even in moderate therapeutic doses. These authors inserted a polyethylene catheter into the subarachnoid space of 19 unselected patients without neurological disease, and observed their cerebro-spinal pressure as it was affected by these drugs. Morphine, 10 milligrammes given intravenously, produced the usual rise, up to 45 millimetres of water on the average, but when it was given thirty minutes after nalorphine, 10 milligrammes intravenously, the rise was much slower and less sustained, and averaged only 15 millimetres of water. Nalorphine alone was capable of producing a rise of pressure averaging 30 millimetres of water, but when it was given thirty minutes after morphine it produced an average fall of 30 millimetres of water.

Simultaneous measurements of alveolar ventilation and alveolar gas composition were made on two patients; concurrently with the rise in intracranial pressure induced by morphine there occurred a fall in alveolar ventilation, an increase in alveolar carbon dioxide content and a decrease in alveolar oxygen content; these changes were reversed by nalorphine. Nalorphine given alone increased ventilation. Voluntary hyperventilation produced a rise in intracranial pressure, and this effect was increased by morphine, but nalorphine neutralized or prevented it. These results support the hypothesis that the increase of intracranial pressure which morphine produces is due to an increase in arterial carbon dioxide tension, with consequent increase in cerebral blood flow, secondary to reduced pulmonary ventilation, and these authors suggest that the antagonistic effect of nalorphine is due to its stimulating effect on ventilation. They also suggest that hyperventilation alone can produce the same reversals of the effect of morphine. This, of course, leads to the control of increased intracranial tension in hypertensive patients who must be given morphine, and supports the conclusion that if they are conscious and can practise hyperventilation they will produce beneficial results, but that if this is not so, nalorphine will produce the same benefit.

ANÆSTHESIA IN THE AGED.

IMPRESSED by the frequency of the comment "He's never been the same since his operation", as applied to aged patients, P. D. Bedford[2] was impelled to investigate the records of 4250 patients over the age of sixty-five. Of these, 1193 had undergone some operation under general anæsthesia during the previous fifteen years. Of these 120 showed some degree of mental deterioration; 29 were completely demented. However, Bedford had some difficulty in making accurate assessments of the part played by their operations in some instances, so in his report he confines his discussion to patients of whom he could say, of his own certain knowledge, that they were mentally normal before operation, and that at autopsy there was no evidence of any cerebral infarction. Under his supervision, then, 251 patients aged over fifty underwent a surgical operation, and 18 (7%) who had been mentally normal before operation suffered from complete dementia immediately afterwards. Typically, they were confused, disorientated and doubly incontinent when they recovered from the anæsthetic, and remained grossly demented until they died, in some cases over a year later.

Since the brain is extremely vulnerable to short periods of vascular insufficiency, and since the aged brain invariably suffers from some degree of defective circulation, then cerebral anoxia, anæmia (in the sense of deprivation of nutrients other than oxygen) or stagnation, or circulatory deprivation due to drugs which depress the respiratory centre or the blood pressure, or metabolic disturbances, are likely to have a very severe effect on the aged. In view of this, and of his experiences with the 18 patients mentioned, Bedford recommends that operations on elderly people should not be undertaken unless unequivocably necessary, that the routine use of premedication must be avoided, and only non-depressant drugs such as paraldehyde should be used; that extra care to maintain blood pressure and hæmoglobin and oxygen levels must be taken, that hypotensive surgery is absolutely contraindicated, and muscle relaxants should be avoided if possible; finally, if post-operative confusion is present, hypnotics may make it worse, or even turn a temporary condition into a permanent one.

There can be no doubting the value of Bedford's recommendations. Moreover, he remarks *en passant*, that many a younger patient, labelled as "neurotic" or worse, might well owe his trouble to a period of cerebral circulatory deficiency during general anæsthesia, and warns that hypotensive surgery, when it is undertaken merely to provide the surgeon with a bloodless field, is impossible to justify in patients of any age. There can be no doubting the value of this observation too.

GASTRIC CANCER AND PERNICIOUS ANÆMIA.

IN spite of quite wide variations in the actual figures published by different observers, it seems to be agreed that there is a significantly higher incidence of gastric cancer among patients suffering from pernicious anæmia than among the general population. N. Zamcheck, E. Grable, A. Ley and Leona Norman[1] have conducted a survey of 1222 patients with pernicious anæmia in four institutions in Massachusetts, and have found that approximately 10% of them developed gastric cancer. They suggest that this high incidence warrants the routine use of screening tests, but they point out that as gastric cancer metastasizes early tests made annually would probably fail to save many lives, so that six-monthly, or preferably three-monthly, examinations are desirable. However, to carry out three-monthly X-ray examinations on all pernicious anæmia patients would be very costly, and would consume much time on the part of specially trained staff. Accordingly they recommend examinations of stools for occult blood and of the gastric contents for cancer cells, which are more suited to the circumstances and are relatively inexpensive and easy to perform. It is a pity that we have no statistics in Australia on the association of these diseases, but since there is no reason to suppose that they would be any different from the American figures, the desirability of adopting some such method of screening may be worth consideration.

PENICILLIN AND RHEUMATIC HEART DISEASE.

A FOLLOW-UP STUDY, after seven years, of patients who had had scarlet fever and had been treated with penicillin gives food for thought. L. Weinstein, N. H. Boyer and M. Goldfield[2] carried out this study of 110 ex-patients; ten of these 110, at the time of their scarlatinal infection, were thought to have developed acute rheumatic fever, because of transient evidence, in particular sore throat and after a latent period increased *P-R* and *Q-T* intervals. All had group A streptococci in the respiratory tract. Seven years later when these patients were examined, the 100 who had not been suspected of having rheumatic fever

[1] *New England J. Med.*, June 30, 1955.
[2] *Lancet*, August 6, 1955.

[1] *New England J. Med.*, June 30, 1955.
[2] *New England J. Med.*, July 7, 1955.

originally still showed no signs of the condition, whereas of the 10 suspects who had had no intervening illness and no chemoprophylaxis, six had definite cardiac lesions and two had probable indications of former rheumatic fever, while two had no abnormalities.

The obvious implication of these data is that although the penicillin treatment markedly suppressed the clinical features of the original attack of rheumatic fever, it nevertheless failed to prevent some degree of rheumatic carditis. Weinstein and his colleagues admit that their findings do not agree with those of many other workers, but insist with reason that their figures are significant, and that it is only after a long-term follow-up that the remote effects of the original modified infection can be ascertained. Because of this they state that a prolonged atrio-ventricular conduction time, appearing after a latent period, in spite of penicillin treatment for a pharyngeal streptococcal infection, must be regarded as possible evidence of modified rheumatic fever—further, it may be the only piece of evidence, and therefore it should always be looked for. A secondary implication is that penicillin may not always prevent the onset of rheumatic carditis, and the oral use of penicillin as a prophylactic may not always give protection. The trouble is that there is no way of estimating how many of the original 102 non-rheumatic patients in this study would have developed rheumatic carditis had it not been for their penicillin treatment, so that this study offers no evidence against the probable value of prophylactic penicillin therapy in some cases at least.

AMŒBIASIS AND RHEUMATOID ARTHRITIS.

THREE years ago Robert E. Rineheart reported having found an unusually high incidence of *Entamœba histolytica* infections among patients with rheumatoid arthritis. Since then he has made an extended investigation, involving more accurate methods of diagnosis, and he has now, in conjunction with Helen Marcus,[1] produced the following results: he considers that examination of a wet preparation stained with iodine, preferably after concentration, is the most sensitive method for detecting cysts of *E. histolytica* in stools; a single examination will find only 40% to 60% of those infected, but when six examinations are made at intervals of two or more days, 95% or more of those infected will be discovered. With this method, a survey of healthy persons and of non-arthritic clinic patients disclosed that approximately 30% of these, living in Oregon, United States of America, are at any one time subclinically infected with *E. histolytica*, while 95% of 150 clinic patients with rheumatoid arthritis had subclinical infections.

In the same journal Dr. Rineheart reports the results of treating patients suffering from rheumatoid arthritis with chloroquine. He chose this drug because previous tests had convinced him that it was the most effective, and at the same time the least toxic, of the available amœbicides. He found that eight of 11 children experienced major improvement or complete remission of the rheumatoid state, and eight of 14 adults experienced a lesser degree of improvement; he remarks that the adults, in view of the duration of their arthritis, actually showed a relatively large degree of improvement. In spite of his findings, however, Dr. Rineheart warns readers that a causal relationship between amœbiasis and rheumatoid arthritis is not proved, and he merely suggests that chloroquine therapy is worth a trial.

In the present state of our knowledge of rheumatoid arthritis, it would be foolish to decry any intelligent attempt at finding a solution to its problems. Nevertheless, it is probably wise to mention here two other reports on this disease. J. J. R. Duthie, M. Thompson, Moira M. Weir and W. Bell-Fletcher[2] have concluded that hospital treatment alone is followed by substantial improvement in the majority of patients with rheumatoid arthritis; this effect compares favourably with what has been claimed for gold or cortisone. It would seem that this factor must be taken into account in any assessment of treatment for rheumatoid arthritis. W. E. Miall,[1] after a survey of men living in a Welsh mining valley, found evidence to suggest that in those with rheumatoid arthritis there was an inherent abnormality of tissue reaction, not confined to the skeletal system. Thus, 46% of those with rheumatoid arthritis also had massive pneumonokoniosis, and 19% had post-primary pulmonary tuberculosis, compared with 19% and 11% among those without. There is no support for the theory that fibrosis or tuberculosis causes arthritis. However, it is not impossible that an abnormality of tissue reaction could, through susceptibility to toxins produced by amœbæ, allow of the development of rheumatoid arthritis. It is to be hoped that Dr. Rineheart's suggestion will be thoroughly tested.

[1] *Northwest Medicine*, July, 1955.
[2] *Ann. Rheum. Dis.*, June, 1955.

THE TAKING OF PENICILLIN ORALLY.

Now that the oral administration of penicillin for the prophylaxis of rheumatic fever is an established procedure, it becomes important to assess the reliability of patients, or of the responsible parent, as regards their following instructions implicitly. A survey on these lines recently made by D. N. Mohler, D. G. Wallin and E. G. Dreyfus[2] has confirmed the impression that many patients are unreliable in this respect. Every general practitioner is familiar with the patient who takes up to two-thirds of an eight-ounce bottle of medicine and then tapers off—most doctors themselves rarely get as far as two-thirds if they happen to be ill. However, Mohler and his associates found that of 245 patients with acute pharyngitis or *otitis media* who were ordered penicillin tablets, no more than 161 took them as ordered; the remainder either felt quite well, or became careless, or could not afford the tablets, or said they made them sick, or else misunderstood the instructions (which were given very clearly and with emphasis). In another survey it was found that when streptococcal infections appeared while penicillin was supposedly being taken by mouth as a prophylactic agent, they usually occurred in such uncooperative patients. This is nothing new, of course, and many statistics which are confidently proffered as proof of this and that are suspect because of possible lack of cooperation, but it is useful to have experimental evidence, and a tangible figure of 34·3% failure to obey instructions on which to base calculations.

THE WORLD-PROBLEM OF MALARIA.

THE World Health Organization has published the report of Dr. E. J. Pampana, Chief of the WHO Malaria Section, and Dr. P. F. Russell, WHO Malaria Consultant, under the title of "Malaria: A World Problem". After outlining the history of malaria and its prevalence and distribution, the authors describe its stunting of physical and mental development, its restricting of social growth, and its blighting of agriculture, commerce and industry. Not only do countries where malaria is endemic face an appalling cost in sickness and death, and an indirect cost in economic loss and inefficiency, but these countries are sources of raw material for others, so that the cost of the disease is reflected in non-malarious countries in the high price of imports. Not only is it true that "no country with a malaria problem can afford not to solve it", but indirectly all countries with even remote connexions with malarious countries have a considerable interest in helping them to solve their problems.

Although much progress has been made in malaria control during the last ten years, usually with help from

[1] *Ann. Rheum. Dis.*, June, 1955.
[2] *New England J. Med.*, June 30, 1955.

WHO, there is still much to do. WHO provides direct assistance; it also provides demonstration teams, pilot projects and advisory officers, and affords a service providing information, advice and training to all who need these things. The final section of this report is concerned with the development of resistance by insects to insecticides, and an important point, not generally realized, is brought out in this connexion. Insect resistance is developing, and there may come a time, perhaps not so far distant, when the world's resources in safe insecticides will run out. If malaria has not been universally controlled before this happens, some countries will have missed the bus—and as well as being condemned to carry their burden of malaria, they well might serve as foci for the re-spread of the disease back to the controlled countries. There is, indeed, a potent argument in favour of every country's taking prompt anti-malarial action on the fullest possible scale, and Australia's responsibilities towards New Guinea in this regard should be emphasized.

THE ENTERIC FEVERS.

THE Royal College of Physicians of Edinburgh has had printed the Sydney Watson Smith Lecture for the year 1954, which was delivered by Adam Patrick. The lecture, entitled "The Enteric Fevers, 1800-1920", is a most interesting account of these diseases, from the stage when typhus and typhoid fever were undifferentiated, through the phases of clinical identification of the "putrid malignant" typhus and the "slow nervous fever" (typhoid) of Huxham (1739). In England, from 1830 on, Perry, Barlow and Stewart described the two diseases, and Jenner clinched the matter. The next phase was clarification of the ætiology and epidemiology of typhoid; here controversy was brisk, and attempts to improve drains met with considerable opposition. The resistance, in Parliament to sanitation Acts, and in *The Times* to the arbitrary control of health by "a sort of sanitary bombailiff", is well described. The development of sanitation and immunization is followed. In print the lecture reads so well that it is obvious that those who had the privilege of listening to Dr. Patrick deliver it must have had a thoroughly enjoyable experience.

DIAGNOSIS OF PHÆOCHROMOCYTOMA.

IN these columns, on June 26, 1954, we discussed the use of "Regitine" (phentolamine) in the diagnosis of phæochromocytoma, commenting on its freedom from side effects and its comparative accuracy. Many writers have given favourable reports, the latest being T. H. Newton, G. I. Smith, F. O. Kolb and D. R. Smith,[1] who describe their use of "Regitine" in an interesting case. *En passant*, they give an assessment of various drugs used similarly for diagnosis, and state that only one false negative response to the "Regitine" test has been recorded so far, but that there have been many false positives. They recommend that all positive results should be checked by other methods before operation is undertaken. In this connexion a paper by R. Moulton and D. A. Willoughby[2] is of interest. These workers have developed a test which, in 250 trials, has produced no false positive or negative results, and which is so simple and rapid that they recommend it for the rapid screening of all patients with hypertension not due to obvious causes. This test, however, is applicable only to large hypertension clinics, or to laboratories which receive large numbers of samples for testing. It depends on the increased urinary excretion of adrenaline and noradrenaline by patients with a phæochromocytoma, of the order of 250 microgrammes or more per twenty-four hours, by contrast with the normal amount of 63 microgrammes or less. The patient passes urine into a sterile jar containing 50 milligrammes of ascorbic acid to prevent oxidation, and the samples are stored at 0° C.

The test is in the nature of a biological assay. Urine is injected intravenously into a cat, and the arterial pressure is recorded on a smoked drum from a recording cannula in the carotid artery. Results are tested against standard solutions of adrenaline and noradrenaline in normal urine, and all tests are duplicated. When positive results occur, further tests are made to eliminate the possibility of error. At least 20 specimens can be tested on one cat. Among 250 patients with hypertension so tested, seven were shown to have a phæochromocytoma. Although this test is not likely to be available to everybody, it would be quite simple to perform it in a laboratory which was adequately equipped, and the certainty of positive results would be very useful. No doubt the establishment of facilities for the carrying out of the test would create a large demand for it.

[1] *New England J. Med.*, June 9, 1955.
[2] *Lancet*, July 2, 1955.

FAT ABSORPTION FOLLOWING GASTRECTOMY.

Loss of weight and excessive excretion of fat in the stools have long been known as sequelæ of total and partial gastrectomy. The severity of the signs varies directly with the size of the portion of stomach removed. R. H. F. Brain[1] lists as the possible causes for this: (*a*) difficulties of ingestion, (*b*) defects of absorption, (*c*) disturbance of fluid balance and (*d*) increased utilization of food. The second of these has the greatest significance. Explanations given for this deficiency in fat absorption are decreased neurogenic and hormonal stimulation of the pancreas, and varying degrees of jejunitis.

A more recent study by M. Courmoulis, E. Gisinger and A. Neumayr[2] indicates that a low serum iron content also plays a part in disturbing fat absorption. These workers investigated 26 patients who had undergone a Billroth II resection and who were found to have normal pancreatic and liver function, as well as minimal changes in their jejunal mucosa. A definite relationship was found to exist in these patients between their serum iron content and fat absorption. Disturbances in fat absorption were greatest in the presence of an iron-deficiency anæmia, but were also present when the serum iron content was reduced in the complete absence of any anæmia. In the latter case the patients showed a gain in weight after administration of iron. Other forms of anæmia in which the serum iron content was normal had little influence on fat absorption.

Iron deficiency may cause a reduction of fat absorption in several ways. In an effort to conserve iron the gall-bladder excretes less bile pigments into the bowel, which renders the emulsification of ingested fats less complete. Iron-containing enzymes such as cytochrome, catalases and peroxydases are also decreased in amount and these in turn affect the activity of the villi adversely. Lastly, when anæmia is established, a relative anoxia of the jejunal mucosa will decrease its absorptive powers.

[1] *Lancet*, May 26, 1951.
[2] *Deutsche med. Wchnschr.*, May 27, 1955.

"PRIMACAINE HYDROCHLORIDE", A NEW LOCAL ANÆSTHETIC AGENT.

A NEW local anæsthetic agent, first synthesized by Epstein and Myer, has been tested by L. M. Monheim,[3] first on rats and then on human patients. It was used, for dental procedures, in a 1·5% solution with adrenaline hydrochloride one in 50,000 to one in 400,000. Anæsthesia was rapidly induced, and with adrenaline at a concentration of one in 50,000 its duration was some thirty minutes. It is not toxic. All this offers little that is new, but the absence of allergic reactions, even in patients with a history of reactions to other local anæsthetics, makes this drug worthy of mention for use in special cases.

[3] *J. Am. Dent. A.*, June, 1955.

Abstracts from Medical Literature.

OPHTHALMOLOGY.

Cyclopentolate Hydrochloride.

D. M. GORDON AND M. H. EHRENBERG (*Am. J. Ophth.*, December, 1954) report on the use of cyclopentolate hydrochloride. The drug is used in drop form in a strength of 0·5% or 1·0%. It was found that it produced wider dilatation of the pupil and more rapid action than homatropine, and that recovery from its effects was complete in twenty-four hours if no miotic was used; recovery occurred in six hours after instillation of 1% pilocarpine solution. In children it has been found that a satisfactory response was obtained in one hour after instillation of two drops of 0·5% solution at five-minute intervals. The drug is free from undesirable side effects, and it does not influence intraocular pressure.

Retrolental Fibroplasia.

N. ASHTON (*Am. J. Ophth.*, April, 1955) gives a review of retrolental fibroplasia. He summarizes his work on the kitten. He states that when kittens of a few days old are subjected to hyperoxia, the vessels growing into the retina are either partially or totally obliterated. When the animal is returned to air after several hours of oxygen exposure, the vessels are unable to reopen, because of intravascular coagulation or adhesion of the vessel walls, and the retina is without an adequate blood supply. A profuse and disordered neovascularization then develops, producing a pathological picture analogous to the early stages of retrolental fibroplasia. There are thus two essential phases in the development of the disease, vasoobliteration in oxygen and vasoproliferation in air. The author maintains that general recognition should be given to the primary importance of oxygen exposure in the genesis of this disease. All efforts should be directed to publicizing this fact in the hope that oxygen will be administered to the premature baby only when essential and then in the minimal amount consistent with the infant's survival.

M. ENGLE *et alii* (*Am. J. Dis. Child.*, April, 1955) have studied the effect of oxygen on premature babies in an endeavour to assess the part it plays in the development of retrolental fibroplasia. They studied 94 premature babies whose birth weights ranged from 800 to 1650 grammes. They found no significant difference with respect to the active or cicatricial phase of retrolental fibroplasia between babies removed from oxygen at two, seven and fourteen days with a four-day weaning-out period and those in oxygen three to four times as long; nor was there any difference in incidence among the babies at concentrations below 40%, between 40% and 50%, and above 50%. The highest incidence of residual changes and blindness occurred in infants with the lowest birth weights, those with a gestational age of twenty-nine to thirty weeks and those in precarious condition in the early neonatal period. They were able to conclude that retrolental fibroplasia does occur in babies exposed to low or moderate concentrations of oxygen for five to ten days.

Precancerous and Cancerous Melanosis of the Conjunctiva.

A. B. REESE (*Am. J. Ophth.*, April, 1955) discusses melanosis of the conjunctiva and its treatment. He states that precancerous melanosis is an acquired lesion, which appears about middle age, has a long period of many years (the precancerous phase) and then appears to be stationary, to progress to a malignant phase, or to regress. It is to be differentiated from diffuse flat nævus and congenital melanosis. It is probable that congenital melanosis is not the forerunner of neoplastic growth. Local excision is not recommended, except for microscopic examination, and in borderline cases the report on the microscopic examination is often indecisive. Although in the precancerous phase the lesion is radiosensitive, the author considers that this is not effective in preventing a precancerous lesion from becoming malignant because of the diffuseness of the lesion. Enucleation is contraindicated, since this leaves diseased or potentially diseased conjunctiva. The author advises exenteration of the orbit when an area of acquired melanosis has shown unmistakable evidence of steady progression and sections of the lesion suggest malignant change.

Surgery of the Subluxated Lens.

D. VAIL (*Am. J. Ophth.*, April, 1955) describes his technique for removal of the subluxated lens. He states that as a rule such a lens is removed by grasping the lens with forceps or erisophake, insertion of a needle into the lens to lever it out, use of a loop or vectis to scoop the lens out and use of support under the dislocated lens to hold it against the posterior surface of the cornea while external pressure is made below. Indications for removal of the dislocated lens are cataractous changes, glaucoma, and uveitis caused by disintegrating lens material. The lens should not be removed so long as vision, either through the periphery of the lens or through the aphakic portion of the pupil, remains good. The author's technique is to insert a Smith spoon into the vitreous and down behind the lens, which is brought forward against the cornea and iris. The lens is expressed by external pressure below. The lens slides out along the smooth surface of the spoon.

Rise of Intraocular Pressure Due to Hormonal Steroid Therapy in Uveitis.

J. LAVAL AND R. COLLIER (*Am. J. Ophth.*, February, 1955) report three cases of uveitis in all of which a rise in intraocular tension developed on systemic administration of ACTH or cortisone. The authors state that these are not to be confused with cases of glaucoma secondary to uveitis, in which systemic steroid therapy often dramatically reduces the intraocular pressure. ACTH does not reduce intraocular pressure in primary glaucoma, and furthermore it does not elevate the intraocular pressure in normal eyes. The authors are of the opinion that systemic steroid hormone therapy promotes the production of a greater degree of intraocular pressure in cases in which there are already changes which hinder normal drainage of intraocular fluid. The increased production of intraocular fluid may occur from alteration in the electrolyte balance among various intraocular fluids, or some reduction of the inflammatory reaction with subsequent improvement of the function of the secretory ciliary processes may be induced by steroid therapy. With patients suffering from uveitis in whom steroid therapy causes an increase in intraocular pressure the simultaneous administration of "Diamox" may reduce the tension to normal limits.

Ocular Molluscum Contagiosum.

B. CURTIN AND F. H. THEODORE (*Am. J. Ophth.*, March, 1955) describe the features of *molluscum contagiosum* as it affects the eye. They state that classical nodules present no diagnostic difficulties, but in the region of the eyelids they may be atypical, resembling a verruca, sebaceous cyst or fibroma. The condition may produce conjunctivitis as well as superficial punctate keratitis. *Molluscum contagiosum* is a virus disease, and transmission is by direct contact. The treatment is local removal of the lesion.

Modern Approach to Glaucoma.

K. L. ROPER (*Am. J. Ophth.*, March, 1955) reviews the salient literature on glaucoma up to September, 1954. The history, classification and mechanism of the disease are discussed, followed by diagnosis and treatment, and finally is considered secondary and congenital glaucoma. The author is of the opinion that the general practitioner should be trained in the use of the tonometer and that the measurement of intraocular tension should be a part of every general medical examination.

Management of Alternating Strabismus.

A. SCHLOSSMAN AND J. SHIER (*Am. J. Ophth.*, March, 1955) have reviewed 230 consecutive cases of alternating strabismus. They found that in 47% of cases the onset of the squint was after the second year. The visual acuity was equal in each eye in the majority of patients; 79% had a preference for one eye. The treatment of alternating esotropia follows the same pattern as for monocular esotropia. In planning surgery the authors are of the opinion that for the average ophthalmogist the recession-resection operation is the procedure of choice. If this does not satisfactorily correct the deviation, operation on the fellow eye can be planned in the light of experience obtained from the first eye. When the deviation for near is greater than the deviation for distance then the lesser deviation should be corrected; the author recommends bifocals to help the residual deviation for near. Patients who measure esotropia and exotropia at different times should

not be subjected to surgery, nor should patients who are intermittently orthophoric and esotropic. In many cases the deviation is psychogenic. When the angle is variable, then the least deviation is the one to correct.

Corneal Transplantation.

R. T. PATON (*Arch. Ophth.*, December, 1954) discusses corneal transplantation and reviews 365 operations, of which 311 were penetrating and 54 were lamellar. In all but one case round grafts were employed. When grafts were six millimetres or less in size, overlying sutures were used; and when grafts were larger than six millimetres, direct sutures were employed. Approximately two out of three grafts remained clear, but the author attributes this high incidence to the fact that a large number of grafts were performed for keratoconus. In 89% of cases of keratoconus there were clear grafts, and in the majority of these a six-millimetre graft was used. The author enumerates the various preoperative diagnoses of the remaining eyes and discusses the results in each group. The final visual results showed an improvement in 72% and no improvement in 20%, and in 6% the results were made worse, in patients who were followed for two months or more.

Herpes Zoster as a Cause of Congenital Cataract.

P. A. DUEHER (*Am. J. Ophth.*, February, 1955) reports two cases of congenital cataract in patients whose mothers had *herpes zoster* during pregnancy. In one case *herpes zoster* appeared in the fourth month of pregnancy, and in the other during the third month of pregnancy. The appearance of cataracts and their response to surgery was similar to those of rubella cataract. One of the patients also exhibited mental retardation, *talipes equinovarus* and microphthalmus. The author regards *herpes zoster* in early pregnancy as the cause of the congenital abnormalities described.

OTO-RHINO-LARYNGOLOGY.

Corticotropin and Cortisone

A. G. RAWLINS (*J.A.M.A.*, February 5, 1955) states that the pituitary-adrenal axis plays an important role in the body's response to stress. Stress stimulates the anterior pituitary lobe to secrete a greater amount than usual of corticotropin, which in turn stimulates the secretion of increased amounts of the cortical hormones. The glucocorticoids cut down on the walling-off process of chronic infection by suppressing all elements of granulation tissue and making the ground substance a better barrier against the spread of early infection. For maximum stimulation of the adrenal cortex, corticotropin should be given intravenously; it should often be used alternately with the glucocorticoids which depress the adrenal cortex. All the hormones should be used with great caution. There are a number of medical conditions in which their use is contraindicated. In severe and obstinate cases of hay fever and in *status asthmaticus* corticotropin gel may be given, in combination with cortisone or hydrocortisone by mouth. Reactions to penicillin and other drugs, angioneurotic œdema and hives are usually well controlled with a combination of corticotropin and cortisone or hydrocortisone. In acute œdema of the larynx, use of these hormones may prevent the necessity of doing a tracheotomy. When histamine cephalalgia and other vascular headaches fail to respond to histamine and other vasodilating therapy, two or three intravenous injections of corticotropin will often bring about improvement. After curettage or fulguration of a contact ulcer of the larynx, the use of corticotropin and the glucocorticoids will frequently prevent further granulations and enhance healing. In fresh stricture of the œsophagus the use of these hormones may prevent fibrosis and lessen stricture formation. After the removal of nasal or laryngeal polypi a course of hormone therapy will aid in preventing recurrence. For eczema and dermatitis of the external ear the local use of a combination of hydrocortisone with the proper antibiotic put up in propylene glycol is very beneficial. In chronic vasomotor rhinitis the hormones are to be used only for temporary aid while desensitization is being carried on. Early treatment of Bell's palsy with corticotropin or cortisone may cut down the œdema of the facial nerve and relieve the condition.

Carcinoma of the Vallecula.

W. F. KEIM (*Ann. Otol., Rhin. & Laryng.*, December, 1954) states that carcinoma of the vallecula may give rise to early and rather characteristic symptoms, and if looked for may be discovered at a time when the disease can be treated with excellent possibilities for cure. The epiglottis may often lie against the tongue and thus may easily obscure the vallecular sulci. Persistent hypertrophy of the lingual lymphoid tissue may also interfere with inspection. Of 342 cases observed, the valleculæ could not be seen in 64 or 18·7%. The only way in which adequate visualization could be obtained was by backward displacement of the tip of the epiglottis under local anæsthesia. A constant soreness in one side of the throat and a vague burning pain on swallowing tart juices or hot foods were the symptoms complained of in two cases described. The lesion in the vallecula was at first missed, but was later discovered when the epiglottis was deliberately lifted forward off the tongue. There were no enlarged cervical lymph nodes. As the lesions were small, it was decided to attempt excision rather than treatment by irradiation. In each case anterior transverse pharyngotomy was the method of surgical approach. A tracheotomy opening was made before the operation for resection of the growth. The operations were planned to preserve both hypoglossal nerves and both superior laryngeal nerves, so that there should be no permanent impairment of swallowing. The choice lay between anterior transverse pharyngotomy and lateral pharyngotomy. The anterior approach was considered the better. The author states that a transverse incision at the level of the hyoid bone extends from one sterno-mastoid to the other. The muscular attachments are severed from the upper and then from the lower surfaces of the hyoid bone, care being taken to identify and hold aside the hypoglossal nerve, and tie the lingual artery. The superior laryngeal nerve is identified and retracted backward; this permits dissection of the greater cornu of the hyoid bone. It is possible next to enter the pharynx through the base of the tongue on either side well away from the vallecula. The corpus of the tongue recedes anteriorly into the mouth. With the nerves isolated, much of the base of the tongue can be resected without compromising the mobility of that organ. The epiglottis and vallecula are easily seen, so that the tissues involved along with the hyoid bone can be removed *en bloc*. The cut edge of the mucosa of the tongue is firmly sutured transversely to the laryngeal mucosa. This is reinforced with a second line of sutures, and the infrahyoid muscles are united with the mylo-hyoid and hyo-glossus.

Heparin in Perceptive Deafness.

E. I. PICK (*Arch. Otolaryng.*, April, 1955) states that little is known about the ætiology of a large group of cases of end-organ deafness, developing around middle age, progressing slowly or rapidly and often accompanied by tinnitus. The lesion may be atherosclerosis. The possible prevention on the basis of atherosclerosis is discussed. Evidence has been accumulating that atherosclerosis is a disorder of fat metabolism in which plasma lipids infiltrate the vascular wall. The lipids in the serum are carried in the form of lipoproteins of varying molecular size. The vascular wall may act as a filter. Some molecules of large size are sifted out and deposited intramurally. The breaking down of lipoproteins from the large to smaller molecules may be due to heparin. In several patients under treatment with heparin for coronary disease a decrease in tinnitus was reported. This observation initiated the present study. Thirty-five patients with perceptive deafness, aged between fifty-three and sixty-six years, were treated. Bleeding and clotting times were checked at the beginning of treatment and once each month thereafter. Blood cholesterol levels ranged between 280 and 350 milligrammes per 100 millilitres. The diets were not altered. The audiograms were checked each month during treatment. The heparin treatment was started with 100 milligrammes given intravenously twice weekly, and later 150 to 200 milligrammes were given twice each week. Intravenous treatment was replaced with intramuscular or subcutaneous injections excepting when hæmatomata occurred. A marked diminution of tinnitus was reported in all cases, and in several that symptom returned with cessation of the treatment. Hearing showed very little improvement, however, even after treatment for as long as twelve to twenty months, although it is thought that with prolonged treatment some arrest of the disease may be expected, and that better results may be obtained in patients treated earlier than those of this series. No further deterioration of hearing was noted in any case during treatment. No toxic reactions, hæmorrhage or other ill effect were noted.

On the Periphery.

MEDICO-HISTORICAL RELICS IN GLASGOW AND ABERDEEN.

THE Hunterian Museum at Glasgow demands consideration separately, but in the lofty, dignified rooms at the Royal Faculty of Physicians and Surgeons there is, in addition to a library of considerable scope, a small museum devoted predominantly to local medical history. The Faculty itself was founded as the result of a charter granted in 1599 by King James VI to Peter Lowe; with the exception of one volume destroyed by fire, the minutes of the Faculty are intact from this date, with, indeed, a version of the earlier volumes in English. The library possesses a first edition of Peter Lowe's "Discourse of the Whole Art of Chirurgerie" (1596), probably the first text-book of surgery in English, and, as a more personal memento, a most ornate pair of his gauntlets. The Faculty was empowered to regulate practice in Glasgow and south-west Scotland, and to this end it undertook the examination of candidates. This led ultimately to an unfortunate and prolonged conflict with the University of Glasgow and its graduates, which was resolved only by the *Medical Act* of 1858, when the determination of fitness to practise became vested solely in the General Medical Council. Among its other duties at one time, as with the London College of Physicians, was to ensure that drugs sold were of a suitable quality.

The museum contains many relics of notable Glasgow graduates and practitioners. The case of surgical instruments carried on his travels by David Livingstone, a Licentiate and later an honorary Fellow of the Faculty, is there, and so is an excellent portrait of the owner. Near by another historic set of instruments on display is from among those used by William Beatty, M.D., on H.M.S. *Victory* at Trafalgar. The ophthalmological instruments and coloured wools which belonged to William McKenzie lie next to several pewter syringes, including some for eye work. Two of Sir William Macewen's sterilizers have been preserved. The relics of Lord Lister include candleholders and nursing utensils from his ward at the Glasgow Infirmary; the fireplace from the ward is in actual use in the museum, and a very fine fireplace it is. The most interesting Lister item is one of a series of letters which have been preserved; in it Lister condemns a "surgical aspirator" of the needle, exhaust pump and bottle variety, invented in about 1869 by W. B. Hilliard, a surgical instrument maker of Glasgow. Lister seems to have regarded aspiration through a needle as impractical if not inadvisable in treatment. The letter is published in *Glasgow Medical Journal*, Volume CXII, 1929, page 95.

Although administratively part of the library, the amount of manuscript material of local historical significance is worthy of mention, and in particular the collection of students' lecture notes and of medical essays written for various purposes; there is also a notebook kept by the industrious William Clift.

At Aberdeen, again it is the library which is of the greatest historical importance, but in the imposing granite building of Mareschal College there is an excellent museum of ethnological, archæological and anthropological exhibits. A good selection of aboriginal Australiana is displayed close by a map of the world on which Australia is clearly indicated by a red ribbon. Apart from a cast of Napoleon's face after death, medical interest centres round the collection of Græco-Roman surgical instruments. This contains lancets, catheters (male and female), needles, an ointment scoop, spatulæ, bone elevators and forceps, probes and a four-leafed vaginal speculum.

In the anatomy department museum there is a series of exhibits relating to the days of body-snatching. Several models illustrate the ingenious devices employed to cheat the resurrectionist of his prize. Sometimes a large granite slab was lowered over the coffin, at others the coffin was covered by an inverted "boat" or a complete bivalved casing of iron trellis work. As even these measures proved ineffective, the building of watchtowers in the graveyards became common, in which relatives kept a nocturnal watch until such time as the body was unlikely to be of much use to anatomists. The most elaborate device was the vault designed for the storage of the body above ground over this vulnerable period, after which the corpse was removed for burial. Inside these circular stone fortresses was a rotating platform; the coffin was inserted through the single entrance and the platform rotated one place. By the time the platform had made a complete circuit the body could be buried without risk of interference. A copy of the regulations for the management of one such vault is displayed. Subscribers were to have free use of the vault, but non-subscribers were liable to a fee of up to twenty shillings. No body was to lie in it longer than four months. Coffins were to be airtight, with no chinks, and made of lead with a pitch lining. "Infectious cases" were to be enclosed in lead or tinplate and the whole soldered. Relevant to this theme is the portrait of Dr. Andrew Moir, whose house was sacked and burned after some dissected remains, carelessly reburied in his yard, were found to be the playthings of dogs and children.

Graveyard relics of a different kind are the exhibits in both these museums relating to the stone cists in which prehistoric man of north-east Scotland buried his dead. Two to five feet square inside, they contain cinerary urns, drinking vessels, flints and skeletal remains. The skeletons are often remarkably well preserved. One, that of a male aged about fifty-five years, shows severe osteoarthritis of the spine, but the jaws carry a well-nigh perfect set of teeth. The skeleton of a woman has been found beside that of a newborn child, even the ossicles of which have been identified.

The historical section of the anatomy museum contains a number of items with personal associations. Among the relics of Aberdeen graduates may be mentioned those pertaining to Sir Patrick Manson, notably his notes (1863) on the lectures of Professor Dyce on midwifery and diseases of women and children, Sir James Cantlie and Sir Arthur Keith. There are various reminders of others such as David Ferriar, John Abercrombie, Charles Skene, James Wylie and Francis Adams, who were associated with the university which has the distinction of establishing the first chair in a medical subject in the United Kingdom.

Clinico-Pathological Conferences.

A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL CONFERENCE was held at Sydney Hospital on April 19, 1955, the medical superintendent, DR. NORMAN H. ROSE, in the chair. The principal speaker was DR. T. I. ROBERTSON, an honorary assistant physician of the hospital.

Introduction.

DR. NORMAN H. ROSE: I should like to welcome to this meeting members of the University and of other hospitals, and also Professor Hart, who has come all the way from California. We hope the professor, whose interest is veterinary, will be able to throw some light on the diagnosis of the disease which afflicted one of his own profession. The speaker today is Dr. T. I. Robertson, who has been given an unusual case in which the field has not been restricted by the sudden demise of the patient. We will ask Dr. Robertson primarily to discuss the case having the information which would normally be available to a physician in the consulting room or at the bedside. There may be some other information about this patient which has not appeared on the printed sheet and which will be made available if he requests it. I should like to present the speaker to you.

Clinical History.

The following clinical history was presented.

This case is different from the type usually presented, in so far as the patient has not died. A definite diagnosis was made during life. The principal speaker is asked to discuss the case as it presented on admission, to suggest what tests might help in establishing the diagnosis, and to discuss how he would manage the case. Further information, if available, will be given during the meeting.

Case History.

The patient was a male, aged forty-eight years, a qualified veterinary surgeon. He was a native of Burma and lived there all his life until his arrival in Australia three months prior to admission to hospital. Fifteen years previously he had an attack of dysentery followed by a fistula-in-ano for which he was operated on. He was quite well from that time until seven years before admission to hospital, when he had an attack of diarrhœa with about 20 loose motions per day. The number of motions gradually decreased, and three months later he was passing three well-formed stools per day.

From then till the time of his present admission he had one or two severe attacks of diarrhœa every month, each attack lasting for a few days. At no time had he had less than three motions per day. He stated that the episodes of acute diarrhœa could be stopped by taking sulphaguanidine tablets and that eating animal fats precipitated the attacks. During acute episodes he had no abdominal pain and no vomiting, but passed a lot of flatus. There was no blood or mucus, and the stools were usually dark yellow in colour. When examined in Rangoon nine months before admission to Sydney hospital, and again at Parramatta one month before, no cysts, ova or pathogenic bacteria were isolated.

Physical examination revealed that the patient was in no distress. His blood pressure was 130 millimetres of mercury, systolic, and 90 millimetres, diastolic, his pulse rate was 90 per minute, and there was no fever. The tongue was of normal texture, moist and clean. No masses were detected in the abdomen, and there was no tenderness. Digital examination *per rectum* revealed no abnormality. In the nervous system all deep reflexes were present, all modalities of sensation were intact, and muscle power was normal. The urine contained no albumin or reducing substances. No abnormalities were found in other systems.

The initial blood examination showed a macrocytic hypochromic anæmia, with 10·5 grammes of hæmoglobin per 100 millilitres of blood, cells slightly deficient in hæmoglobin, marked macrocytosis, slight anisocytosis and polychromasia, and numerous target cells.

DR. T. I. ROBERTSON: I should like as a first mode of attack to accent the points which I regard as important in this history. Firstly he is a male, forty-eight years old, and a veterinary surgeon. We are told he is a native of Burma. He had dysentery fifteen years ago with the development of a fistula-in-ano. There was then freedom from illness for eight years. For the last seven years he has had diarrhœa constantly. Diarrhœa has been lessened by sulphaguanidine and was possibly increased by animal fats. On two occasions, about two months apart, the results of stool examination were bacteriologically negative.

The important things to my mind are the locality (tropical residence), his occupation, the history of diarrhœa or dysentery for fifteen years with a gap of eight years, the fact that the diarrhœa was affected by sulphaguanidine and animal fats, and the negative findings from stool examinations.

Physical examination reveals a normal tongue, negative findings from abdominal and rectal examination, a normal central nervous system and an abnormal blood count. It might be worth indicating now that the blood count shows macrocytosis which is rather marked for the degree of anæmia, and there does appear to be evidence of iron deficiency as well. In this patient the target cells are probably not indicative of Cooley's anæmia, but are simply large cells deficient in iron.

The problem can be further crystallized into a consideration of the differential diagnosis of diarrhœa with macrocytic anæmia in a man of this type. A number of possibilities come to mind, which I propose to enumerate briefly. Then I wish to talk about some of the more likely ones in more detail and see if we can approach the diagnosis through the history alone.

Amœbic with bacillary dysentery and colitis deserve consideration; also Crohn's disease, steatorrhœa or jejuno-ileal insufficiency, and its many causes; structural intestinal abnormalities such as strictures, anastomoses and blind loops; and infestations, of which lambliasis and *Diphyllobothrium latum* are the most likely. The various causes of macrocytic anæmia also warrant discussion. When one is confronted with macrocytic anæmia, I think the first question is whether or not it is megaloblastic—that is, whether or not there are megaloblasts in the bone marrow. To answer that question is important. Of the megaloblastic types, pernicious anæmia does not appear likely. The group of non-Addisonion macrocytic anæmias contains some likely diagnoses, and we again encounter steatorrhœa and intestinal strictures as well as defective nutrition and liver disease.

The first thing is to consider the diarrhœa or dysentery in greater detail. He had an attack which is called dysentery fifteen years ago, and it was followed by a fistula which required operation. Dysentery is a term often used loosely. We must be prepared to accept that the attack was either amœbic or bacillary dysentery, or simply a severe diarrhœa. The fact that fistula-in-ano occurred may be coincidental—it is not unheard of in any type of severe diarrhœa—but tuberculosis, amœbic dysentery and Crohn's disease are all noted for causing fistulæ. The fact that there is an eight-year interval of freedom is in favour of the original attack being not relevant to the symptoms of the last seven years. However, intestinal tuberculosis may occur as an acute attack, to be followed by healing, an interval of freedom and then diarrhœa due to the formation of anastomosis or stricture. Crohn's disease is capable of healing with subsequent relapse or recrudescence; so again we cannot exclude it at this stage. Amœbic dysentery is well known to be capable of following almost any course and can show very few signs; the patient can live almost in symbiosis with the amœbæ, and symptoms can be very few. Hence, considering this first episode, although there is a possibility that it is entirely irrelevant, amœbic dysentery, tuberculosis and Crohn's disease deserve further consideration, because of the accompanying fistula-in-ano.

After eight years, diarrhœa again occurred with twenty motions each day. It is hard to explain that by anything other than infection or possibly steatorrhœa. The factors given in the history notes said to alter the diarrhœa need thought. The attacks can be precipitated by animal fats, or it is thought that they may be. This is in favour of steatorrhœa. Their cessation under sulphaguanidine therapy seems to indicate an infective origin, but even amœbic dysentery, not directly amenable to sulphaguanidine, may be affected that way, and this has to be regarded cautiously. The fact that there is no abdominal pain, no blood or mucus and no pathogens on two examinations tends to weigh against dysentery and Crohn's disease. The passage of flatus and bloating are points in favour of sprue and its near neighbours. The anæmia, macrocytic and slightly deficient in iron, is highly important. With these preliminary thoughts we might consider the case in more detail.

First of all, he is a veterinary surgeon, but to my knowledge there is no disease to which a veterinary surgeon is exposed which would fit this picture. I think chronic bacillary dysentery can be ruled out because bacillary dysentery probably never becomes chronic and there is usually abdominal pain, tenesmus, blood and mucus in the stool. I think we can exclude it without further ado. Ulcerative colitis can be considered only to be dismissed. I cannot imagine this disease occurring without pain, blood or mucus, or tender colon. Its associated anæmia is almost always hypochromic and deficient in iron. Very occasionally fat absorption can be interfered with, and one can encounter macrocytic anæmia, but it is uncommon. I would be inclined to exclude it. The more innocent forms of colitis are only variants of either mucous or motor colitis, in which either the secretory or the motor functions are accentuated. He had no tender colon, no abdominal pain, no excessive amounts of mucus, and they can be dismissed. Amœbic dysentery is a much more likely diagnosis. It may be very chronic. It is possible for infestation to occur and the patient to have no symptoms. Stools can be normally formed. There need not necessarily be a lot of diarrhœa, but it is capable of causing attacks of diarrhœa. Alternating diarrhœa and constipation is quite common. Bacteria in the bowel apparently influence the course of amœbic dysentery, and it is possible, by attacking the bacteria in the bowel, to alter the course of amœbic dysentery; sulphaguanidine may do just that. However, abdominal pain is common, and tender colon is common. I think the diagnosis must rest on the demonstration of amœbæ in the stools. In a person with diarrhœa and long tropical residence amœbic dysentery has to be considered very seriously and amœbæ searched for diligently before the disease can be excluded. Some authorities try the therapeutic effect of emetine on the symptoms before they will consider this diagnosis excluded. One point against amœbic dysentery, and strongly against it, is the macrocytic anæmia. The anæmia of amœbic dysentery is usually hypochromic, due to blood loss. However, it is possible for amœbic dysentery to be complicated by coincident malnutrition or hepatic disease, which can cause macrocytosis.

Crohn's disease deserves further consideration. It may occur in acute, subacute and chronic stages. Both acute and subacute stages and rarely chronic stages can heal. This patient could have had Crohn's disease fifteen years ago, associated with fistula, which healed only to relapse eight years later. Diarrhœa can be of sudden onset. Malabsorption of fat can occur simply because a lot of the small intestine may be affected by the disease. Abdominal pain is variable. It can mimic idiopathic steatorrhœa perfectly. Blood in the stools is uncommon in Crohn's disease. There are many points in favour of its being the correct diagnosis in this case. Points against it are, however, strong ones. Long-continued Crohn's disease usually shows predominant abdominal pain. There is usually a fever, acute abdominal episodes, abdominal masses, sinuses and fistulæ between the gut and abdominal wall, between

the segments of the gut and between the gut and other organs. Barium studies will help in its recognition. It cannot be dismissed yet.

One of the various types of steatorrhœa could explain this illness characterized by diarrhœa, lassitude, loss of weight and glossitis. The anæmia is often macrocytic, sometimes megaloblastic, and the degree of macrocytosis is at times high, even when the anæmia is not very marked, as in this man's case. It occurs in the well-to-do more often than in the poor natives. Bowel habits often are not very abnormal; diarrhœa may come in attacks. Flatulence is common, and so is the passage of flatus. Neurological signs are uncommon, and so is the passage of flatus. Neurological signs are uncommon. There are various other positive points which help in diagnosis: clubbing occurs in about 15% of cases, calcium and vitamin malabsorption result in bony defects, and pigmentation is common. These things we do not know. Pancreatic disease is another cause of steatorrhœa; when there is any long-continued diarrhœa without gross anæmia or glossitis, pancreatic disease should be considered. However, it does not appear capable of explaining the whole picture. Again, pancreatitis or pancreatic fibrosis can occur in some of the malnutrition syndromes, as, for instance, in kwashiorkor, where there is hepatic cirrhosis and pancreatic fibrosis, iron deficiency anæmia and extreme malnutrition.

Intestinal tuberculosis should be considered. The hypertrophic type forms a mass, which has not occurred here. The ulcerative type is capable of healing and of causing stenosis, or anastomoses, which can in turn cause macrocytic anæmia and diarrhœa. It can cause extensive lacteal involvement and involvement of the mesenteric glands, with consequent disturbance in the absorption of fat. It may be associated with fistula-in-ano. It must still be kept in mind. Intestinal strictures and anastomoses are mostly due to tuberculosis; some are due to Crohn's disease, some to adhesions. The anastomoses in particular may be associated with diarrhœa, and in many cases the anæmia is macrocytic. It is then possible for an acute attack of diarrhœa to be caused by intestinal tuberculosis, later healing and, later still, subsequent cicatrization or anastomosis between loops of gut, causing chronic diarrhœa.

Liver disease is another possible, although unlikely, explanation of the many symptoms. Macrocytic anæmia may occur, but without malabsorption, malnutrition or pancreatic disease I do not think it can cause the full picture. Therefore, at this particular stage in the discussion I should like to limit the diagnostic possibilities to steatorrhœa or, perhaps more broadly, jejuno-ileal insufficiency, Crohn's disease and chronic amœbic dysentery.

Now, I feel that the people who took the history would be in possession of more facts than I am at the moment. I am denied the very important point of being able to look at the patient. There are some questions which, with that history, I would ask the patient, and some signs I would look for before I went on with special investigations. These questions may influence my subsequent thoughts. If it is reasonable, I would like to ask them now.

We are told he was a native of Burma. He is a dark-skinned man; is that right?

DR. G. MICHELL: Yes.

DR. ROBERTSON: The reason I ask that is that a native of Burma is different from a Burmese native. In speaking of macrocytic anæmias it is quite important, because pernicious anæmia is practically unheard of in people of non-Nordic descent. What has his diet been like, and does he drink alcohol?

DR. MICHELL: The notes say he drank alcohol occasionally and that he ate a normal Burmese diet, whatever that is, until he arrived in Australia.

DR. ROBERTSON: He was a man practising as a veterinary surgeon, presumably of high social class. Has he lost weight?

DR. MICHELL: There is no mention of his present weight, but it was stated that he lost two pounds during episodes of diarrhœa and regained it shortly afterwards.

DR. ROBERTSON: What is his general condition at the moment?

DR. MICHELL: It is quite good.

DR. ROBERTSON: Did he have a protuberant abdomen?

DR. MICHELL: No.

DR. ROBERTSON: What did the ano-rectal scar, if any, look like?

DR. MICHELL: There is no note about that.

DR. ROBERTSON: I ask that because if it is tuberculosis, it is conceivable that there might be an ugly scar, as in ulcerated tuberculous cervical glands. Are there any comments about his teeth, bones or fingers clubbing?

DR. MICHELL: There was no clubbing. His teeth were described as perfect, and his bones seemed to be quite normal.

DR. ROBERTSON: Is there any family history, especially of anæmia or tuberculosis?

DR. MICHELL: It was specifically mentioned that there was no tuberculosis in the family.

DR. ROBERTSON: Were there any notes of past history, operations, accidents and the like?

DR. MICHELL: No. The only other remark is concerning the fistula, and the only other disease mentioned is the attack of dysentery.

DR. ROBERTSON: I ask that because gun-shot wounds and intestinal operations can cause a condition like this. I would inquire carefully about any illness he had fifteen years ago to find whether he might have had tuberculosis then. His stools, I note, were yellow. What about odour and consistency?

DR. MICHELL: There is no note about odour. During attacks they were said to be loose and pale, and at one stage frothy. There was no mention of amount. In the intervening periods they were of normal colour.

DR. ROBERTSON: At what time of day did he have the looseness of his motions?

DR. MICHELL: There is no record of that.

DR. ROBERTSON: Steatorrhœa tends to cause early morning diarrhœa. Other observations about the stools are important—the presence of blood, mucus or fattiness. What sort of treatment has he been given? I do not wish to know what he has been treated for! I mean what anti-anæmic treatments has he received?

DR. MICHELL: As far as we know, he had had none up to the time of the present admission.

DR. ROBERTSON: Thank you. Historically, although not all points are consistent, I would favour jejuno-ileal insufficiency, namely, sprue, as being the likely diagnosis. I would not, however, completely exclude chronic amœbic dysentery at this stage, or Crohn's disease or intestinal abnormalities such as stricture, anastomoses and the like. I would feel inclined to dismiss bacillary dysentery, ulcerative colitis, pancreatitis, pancreatic disease and chronic liver disease. My own particular approach from this point would be firstly to examine his lower bowel by sigmoidoscope as part of the physical examination. The next thing that I would like is amplification of the blood count, especially absolute values, to try to sort out the question of iron deficiency; and then sternal marrow to answer the extraordinarily important question as to whether the anæmia is megaloblastic or normoblastic. Stool examination would be required again; and this, I might say, does not mean just casually examining the stool, but also taking a swabbing at the time of sigmoidoscopy if there is any abnormality of the colon, looking for amœbæ on a warm stage within minutes of taking the specimen, and looking for cysts and ova on more than one occasion. It has been calculated that only 25% of cases of amœbic dysentery will be diagnosed on a single casual stool examination. If the result of this is negative, it is common current practice to examine six consecutive stools. Knowing the results of sigmoidoscopy and chest X-ray examination, and more about the blood count and marrow and stool, I would take stock. My ideas might be altered considerably by these tests; I do not know.

A second bank of tests, some of which would be necessary, are barium studies of the intestinal tract, fat tolerance test for examination of the stool, examination for occult blood, gastric test meal examination and X-ray examination of bones. I am less enthusiastic about X-ray examination of the bones now that I have been told his teeth are normal and there is no evidence of bone disorder, because these are points against malabsorption of calcium and suggest that the X-ray findings will be negative. The only other things I think of at the present are the questions of pancreatic function tests, liver tests, glucose tolerance test and a further check on absorption from the intestinal tract and possibly a blood calcium estimation. I have been asked to present things in that way. I reiterate that at the present moment I would like to know more of the blood count, sigmoidoscopic findings, chest X-ray appearances and stool examination findings.

DR. ROSE: I think at this stage it would be fair to present the speaker with more information. Perhaps we will not

disclose all the results of special tests until after the audience has had a chance to make its comments, but I think we could give the results of the first battery of tests he mentioned. Dr. Tebbutt has the results of the hæmatological examinations.

DR. TEBBUTT: I have not got a great battery of results. This is what I have . . .

DR. ROSE: Perhaps Dr. Robertson would prefer to question you. You might let the cat straight out of the bag.

DR. TEBBUTT: I do not think I will. The cat is not with me. I have the result of a blood count: red cells 3,950,000 per cubic millimetre, hæmoglobin value 10·5 grammes *per centum*, hæmatocrit 40%, mean corpuscular volume 101 cubic microns, mean corpuscular hæmoglobin 27γγ, mean corpuscular hæmoglobin concentration 26%. Examination of a stained smear showed marked macrocytosis with slight anisocytosis and polychromasia. The reticulocyte count on another occasion was less than 1%. That is all I have.

DR. ROBERTSON: Was the sternal marrow examined?

DR. TEBBUTT: No, I have no marrow.

DR. ROBERTSON: The absolute values confirm the presence of macrocytosis and probable iron deficiency, the mean corpuscular hæmoglobin concentration being only 26%. It bears out, I think, my suggestion that the target cells are large iron-deficient cells. The fact that there is iron deficiency on an allegedly good diet directs attention to malabsorption again, but also to the necessary for excluding intestinal bleeding. We need occult blood examination for this. I think that the omission of sternal marrow examination in macrocytic anæmia is a serious one, because in most cases of sprue the marrow is megaloblastic. Most macrocytic anæmias are megaloblastic, and the causes are fairly limited. I personally would regard this test as of high priority, but we must try to get along without it. Sigmoidoscopy?

DR. MICHELL: No lesions were seen. There is no note made as to how far the instrument was passed.

DR. ROBERTSON: This excludes conditions such as ulcerative colitis and amœbic dysentery, but not Crohn's disease. Most cases of amœbic dysentery do show sigmoidoscopic abnormalities. They may be small lesions and easily missed, but we must take the sigmoidoscopic findings as negative. I think, myself, I would have taken a swabbing at the time of the examination and submitted that for bacteriological examination.

DR. MICHELL: I am sorry I forgot to mention that that was done, and no pathogens were found.

DR. ROSE: Perhaps at this stage we should throw the case open for discussion. Dr. Calov has just come back after spending six weeks in New Guinea. With the medical knowledge he must have acquired, I wondered whether he would open the discussion.

DR. W. L. CALOV: Well, Mr. Chairman, I could talk with great enthusiasm about New Guinea. This case has got me completely puzzled. As a matter of fact, just between ourselves, I think it might have Dr. Robertson puzzled, too! There are two or three things in the clinical examination I should like to know about. For example, we have heard that his belly was not swollen, but there is no mention of his liver. They say no masses were palpable. In sprue—if indeed this is sprue, and it could be—you do not usually even find any liver dullness. Can you tell us anything about that?

DR. MICHELL: The notes merely record that the liver was not palpable.

DR. CALOV: That is one thing. The other is that we do not know the leucocyte count. That might be of interest if there is any possibility of amœbiasis. Dr. Robertson has been separating amœbiasis from sprue. Well, I should be inclined to think of them both in spite of the failure to find any amœbic cysts or ulceration in the rectum or lower part of the sigmoid colon. Amœbiasis and sprue are not an uncommon combination. So I think it is quite possible that this man has both diseases. You cannot rule out amœbic dysentery on failure to find cysts at two examinations of the stools. As for sprue, we certainly have not got enough information here to make the diagnosis. I should like to know more of what the stools looked like, microscopically I mean, and what the results of chemical tests were, also the serum calcium content and one or two other things. I should think that amœbiasis and sprue combined are likely diagnoses. One thing which is strongly against sprue is that the man has not lost weight. I should like to know more about the liver.

DR. ROSE: Could we have the opinion of other members of the audience? Dr. Ritchie, would you care to say something?

DR. F. L. RITCHIE: Dr. Rose, I agree with Dr. Calov. I would be inclined to feel, myself, that the most likely diagnosis is of the steatorrhœa group. I think that the real problem which confronted the clinician is whether this is idiopathic steatorrhœa, in which one can find no underlying cause at all, or whether it is symptomatic steatorrhœa—let us say, tuberculosis of the lacteals—or one of half a dozen much more rare types of illness which, by and large, require a great deal of deep investigation before coming to a satisfactory conclusion. Whether or not he has amœbic dysentery as well has also to be decided. This can be resolved in one of two ways: either by continuing examining his motions for entamœbæ or by treating without proving as Dr. Robertson suggested. To return to the question of steatorrhœa. From a purely clinical point of view I think the very first test I would have had done would be a fatty analysis of stools. If, in fact, the stools show an increase in total fat, then one must be dealing with steatorrhœa. Then one can go ahead from there to decide whether the condition is idiopathic or symptomatic and treat accordingly.

There is one other possibility not mentioned by Dr. Robertson, which is a clinical entity, even if not a real entity. There are a group of people who have had either amœbic or bacillary dysentery, even though treated satisfactorily, whose colons may remain very irritable. This is a very simple explanation, which could be called a post-dysenteric irritable colon. I do not think it is likely in view of the macrocytic anæmia, but it is a condition worth keeping in mind generally.

DR. ROSE: Will you fire your second salvo, Dr. Robertson?

DR. ROBERTSON: I should like the results of biochemical tests.

DR. WARDLAW: A fat balance study was made over a four-day period. The patient was given a diet containing 70 grammes of fat per day—that is to say, a total of 280 grammes over the period. The output of fat in the fæces during the period was 38 grammes, of which 26 grammes consisted of split fats and 12 grammes were unsplit. This gives a fat absorption of 86·5% of the fat ingested.

DR. ROBERTSON: It is usually taken that anything over 90% is normal, but the very lowest limit is 85%. So we can take this as on the borderline. What was the total weight of fat put out?

DR. WARDLAW: Thirty-eight grammes in four days.

DR. ROBERTSON: I would have thought that that might have been a little low, and I would, perhaps, have been suspicious of the test. I mean all these stools have to be taken from the ward to the laboratory, and there is danger of error.

DR. WARDLAW: Well, we analyse everything we get.

DR. ROBERTSON: Were there any other examinations of fat in the stools?

DR. WARDLAW: There was one examination of a random specimen. That gave a total of 34% fat of the stool, and the split fat formed 15% and the unsplit 19%.

DR. ROBERTSON: They are abnormal figures. Correct me if I am wrong, but on a single example like that it is possible to diagnose malabsorption. Is that so?

DR. WARDLAW: Yes, I think so.

DR. ROBERTSON: It is a useful screening test before going on to more detailed fat balance studies. I think there is fat malabsorption, though I would prefer to see a figure below 85% absorption. On the other hand, he has been going for a long time without extraordinary symptoms except the anæmia, and it is possible that this minor degree of malabsorption may fit the picture well. Was the stool examined microscopically for muscle, fat and so on?

DR. WARDLAW: Not in the department of biochemistry.

DR. MICHELL: There is no record of its having been examined in that way. Bacteriologically no cysts, ova or pathogens were seen.

DR. ROBERTSON: Were any other estimations made—for example, calcium?

DR. WARDLAW: The calcium level of serum was 10·2 milligrammes per 100 millilitres and the inorganic phosphorus was 3·6.

DR. ROBERTSON: They are normal figures.

DR. WARDLAW: Yes. Serum alkaline phosphatase was 9·6 King-Armstrong units, also in the normal range.

DR. ROBERTSON: Because we are still searching for malabsorption, was a glucose tolerance curve done?

DR. WARDLAW: Yes. The fasting blood sugar was 117 milligrammes per 100 millilitres, and then the various half-hourly figures were 133, 137, 124 and 109 milligrammes per 100 millilitres of blood.

DR. ROBERTSON: I would regard that as a low curve. To have less than 40 milligrammes rise in the glucose tolerance is usually taken to indicate poor absorption of glucose. Taking this with the abnormal fat content of stools and the borderline fat tolerance, I think we can say this patient had steatorrhœa. So far as causes go, there are sprue, idiopathic steatorrhœa, cœliac disease (which does not apply here), Crohn's disease, intestinal tuberculosis and other intestinal abnormalities. It would probably be relevant to ask now for X-ray studies of the intestinal tract.

DR. MICHELL: No abnormalities were seen in barium-meal examination and follow through. The pattern of the small bowel was normal. There was fairly rapid passage of the meal. Barium enema examination showed a free flow of barium as far as the caecum and then into the small intestine. No abnormality was seen.

DR. ROBERTSON: It would be nice to know that screening was done at four, six and eight hours after the meal, and whether non-flocculating barium was used. It probably has not been, but it shows the pattern much better. It would be nice to know whether the radiologist had been looking specifically for intestinal anastomoses, which are very hard to find sometimes. But we have no radiologists here to answer that. Were examinations made of the fæces for occult blood to test that possible cause of iron deficiency?

DR. MICHELL: No.

DR. ROBERTSON: Well, we are left with an iron-deficient anæmia, which is not explained by deficiency in diet, possibly explained by malabsorption, and we do not know whether there is any intestinal bleeding. I feel that more positive information could have been derived from some of the tests. On what we have I think the diagnosis is steatorrhœa and, more specifically, sprue. I would like to search further for tubercle bacilli, although admittedly intestinal tuberculosis in an adult without other evidence of tuberculosis is uncommon. I would look further for possible intestinal abnormalities. Small intestinal abnormalities can cause this picture, but I think probably the diagnosis of tropical sprue is the correct one.

DR. CALOV: I should like to ask Dr. Wardlaw whether the test of the stools was made during an attack of diarrhœa.

DR. WARDLAW: I am afraid I do not know.

DR. ROSE: Are there any other tests, the results of which have not been disclosed?

DR. MICHELL: No.

DR. ROSE: Dr. Billington has been appraised of the diagnosis of this patient, so I would ask him to sum up and tell us the whole story in the few minutes that are left to us.

DR. B. P. BILLINGTON: It would appear on the evidence given that Dr. Robertson is correct in his diagnosis of sprue. There are some features which are somewhat unusual, a lot depending, of course, on what the X-ray films did in fact look like and whether the small bowel pattern was specifically looked for.

Dr. ten Seldam can correct me if I am wrong—I understand sprue to be a word applied originally to the oral lesion of the idiopathic steatorrhœa syndrome, and this case then would be a case of sprue without sprue. However, sprue generally implies idiopathic steatorrhœa, and there may be two particular varieties, tropical or non-tropical, and one must presume that this was a case of tropical sprue.

There are factors in differentiating between tropical and non-tropical sprue quite apart from the symptom complex. The tropical sprue patient when taken into a non-tropical area may relapse once, but then never relapses again unless the patient goes back to a tropical area which is endemic for sprue. On the other hand idiopathic steatorrhœa may continue for as long as the patient lives, with exacerbations and remissions. In fact it has been said that patients with tropical sprue do not die in temperate zones, but those with idiopathic steatorrhœa may do so. Therefore distinguishing the two conditions from an epidemic point of view may be easy on the grounds of where the patient came from; but if this is not known, then the only point of difference is based on prognosis—tropical sprue patients get better, the others keep on going. It is on record, of course, that there are cases of non-tropical sprue which come from tropical zones, but these are usually in white people.

The failure to find any abnormality in small bowel pattern, if that was so, is unusual in both varieties of the sprue syndrome. I think the majority of people would say there is a characteristic pattern, the so-called deficiency pattern, if one uses flocculating barium; and even if one uses non-flocculating barium, one is likely to find marked dilatation of the small bowel, and this will be present whether the patient is in clinical remission or not. However, approximately 10% of patients who have the sprue syndrome will not show, at any one time, the abnormal radiological features of the small bowel, although they may do so on a subsequent occasion.

A question on steatorrhœa which I think was raised by Dr. Calov was whether the patient had diarrhœa or not at the time the fæces were examined. I think this is a good point. There is no doubt whatever that when patients with this syndrome have diarrhœa with their steatorrhœa, they may in fact have more steatorrhœa. When they are in remission with regard to diarrhœa, they will still have excess fat in their stools, but not to such a great degree. So if one can observe a patient during the diarrhœal phase of the disease, the diagnosis of steatorrhœa is more obvious.

I thought it might be of interest to mention some of the recent work on the amount of fat absorbed over a four-day period. It has been shown that one does not have to have a full fat balance, in the sense that one has to measure the intake and compare it with the output to diagnose steatorrhœa; but provided the diet contains between 50 and 150 grammes of fat per day, any excess of fat in the stools over seven grammes per day is definitely abnormal. There are cases on record where no fat was given by mouth and yet seven or more grammes were excreted in the fæces. This is unusual and unexplained.

As far as the anæmia is concerned, the story fits in perfectly with sprue. Iron deficiency and macrocytosis with or without megaloblasts are typical. All cases, I understand, of the sprue syndrome with anæmia are in fact iron-deficient. The reasons for this are not fully worked out. Some may be in part due to bleeding, due to low prothrombin levels because of poor absorption of fats and fat-soluble vitamin *K*. Most are associated with failure in absorption of iron as part of the general defect in intestinal absorption. But studies with radioactive iron have shown that these two explanations alone cannot account for the iron-deficiency anæmia in some cases, and there must be some other disorder of metabolism in these people where iron given intravenously is not fully utilized. The general feeling is, at least in non-tropical sprue, that this is partly a general disorder of metabolism which has inheritable characteristics.

This particular question in tropical sprue is unanswered, but it is well known that in both tropical and non-tropical sprue there are precipitating factors both for the individual and, in fact, for the area in which the individuals live. Frazer, of course, has postulated that rancid fats are a precipitating factor in tropical sprue, which may well fit this case—rancid in the sense of over-cooked fat with a large proportion of short-chain fatty acids.

One might draw an analogy to the relationship between wheat gluten and cœliac disease in children.

As far as the megaloblasts are concerned, interesting observations have been reported from the use of radioactive cobalt incorporated into vitamin B_{12}. Patients with the sprue syndrome failed to absorb B_{12}, but this was not overcome by giving intrinsic factor. The defect appeared to be in the intestinal absorption of the B_{12} complex. Estimation of the serum levels of B_{12} have shown that there are two types of cases. One type has a low level of B_{12} and responds to treatment with B_{12}, but the response is not universally complete. The other type has a normal B_{12} serum level, and the anæmia responds only to folic acid therapy and not to B_{12}. In one of these latter cases reported by Mollin the patient responded to folic acid in this way, but three years later developed subacute combined degeneration of the cord with a low serum B_{12} level. Perhaps this implied that there was an underlying deficiency of vitamin B_{12} as well.

I have no time left for further comment, but I understand that the present patient was given a therapeutic trial of treatment with emetine and carbarsone without effect. He improved on a low-fat, low-starch diet, and the anæmia responded to folic acid. On discharge from hospital he was free from diarrhœa, and his hæmoglobin value was 12 grammes per 100 millilitres of blood.

Diagnosis.

Sprue, with idiopathic steatorrhœa.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on June 15, 1955, at the Medical Society Hall, 426 Albert Street, East Melbourne, DR. H. G. FURNELL, the President, in the chair.

Atomic and Thermo-Nuclear Warfare.

MAJOR-GENERAL F. KINGSLEY NORRIS, C.B., C.B.E., D.S.O., E.D., M.D., read a paper entitled "It Could Happen to Us" (see page 397).

DR. W. OSTERMEYER asked what the cobalt bomb was.

COLONEL W. D. REFSHAUGE, Assistant Director of Medical Services,[1] said that theoretically the danger from such a bomb lay in the fact that radioactive cobalt had a very long half-life, but so far as he was aware, such a bomb had not been made.

DR. R. F. COLAHAN asked, in the event of an atomic explosion when a doctor outside the immediate danger area was called upon to treat casualties, whether there was any contraindication to using standard first-aid treatment, or whether some special form of treatment would be desirable.

Major-General Norris, in reply, said that while radiation after an atomic explosion increased the hazards, most victims would require immediate treatment for burns and the effects of blast, and standard first-aid treatment was indicated in such cases.

MAJOR-GENERAL SIR SAMUEL BURSTON said that, as medical adviser to the National Headquarters of the Red Cross Society and as Chief Commissioner for Australia of the Saint John Ambulance Brigade, he had given considerable thought to the problem of civil defence. The public attitude was that the whole thing was so dreadful that it was futile to do anything or make plans; but he thought that that attitude was wrong, and one to be combated by the Federal and State Governments. Five years previously he had discussed the subject in the Rupert Downes Memorial Oration, and since that time the problem had become greater, with the increase in the range and size of aeroplanes and the development of super-submarines which could fire shells with atomic warheads. Australia was a major production centre for the anti-communist world in the Pacific and, therefore, a good target for an enemy. Sir Samuel Burston went on to say that four years prior to the meeting, after a combined meeting of representatives of the Red Cross Society and the Saint John Ambulance Brigade, he had prepared a plan, which was submitted without result to the Federal Government, designed to cover such an emergency with reference to equipment, blood substitutes *et cetera* to deal with mass casualties. In the event of an emergency, material, equipment, therapeutic aids, trained personnel and transport would be essential, and necessary preparations could not rapidly be made. It was important to realize that after an atomic explosion there were two problems, which were to some degree separated—first, the immediate results of the explosion, burns, blast effects *et cetera*, and later, the problems of radiation sickness. Those within a mile or so of ground zero would die, and it was at the periphery that the challenge lay to the medical services, civil and military. Radiation effects would not be an immediate therapeutic problem in those who would survive, and, therefore, proper organization to give immediate aid to casualties could save thousands of lives. If no such organization existed, there would be a repetition of what happened in Japan—the nation would "throw in the towel".

Major-General Norris, in supporting the views of Sir Samuel Burston, referred to the fact that the effects of the two atomic bombs on Japan were far more widespread than the resulting local devastation; it was the first time in history that a country had surrendered with its army intact. He said that much could be done on the periphery, and in contradistinction to other disasters, the correct procedure after an atomic explosion would not be to rush to the centre of the damage. The problem was primarily a community problem, not a medical one, and with proper civil training many lives would be saved.

Dr. Furnell, from the chair, said that the scope of the lecture had not left much room for questions. In thanking Major-General Norris for his interesting and valuable address, Dr. Furnell referred to the fact that this was one of the last occasions on which General Norris would appear as Director-General of Medical Services. He had always been a stalwart member of the British Medical Association, a member of the Council of the Victorian Branch for many years, and in times past its honorary secretary and president. Dr. Furnell took the opportunity of saying how much the Victorian Branch appreciated Major-General Norris's services and regretted his retirement from the Council, and assured him that he would carry into his retirement the thanks of all for his valuable work.

Major-General Norris, in reply, said that after a similar lecture in Sydney he had received a letter in the following terms: "Dear General, Either you or Bertrand Russell are lying—I believe it is you." What he and his colleagues had done that night was to tell the truth about the problem, which concerned everyone, and about which something could be done.

[1] Since this meeting was held, Colonel Refshauge has been promoted to the rank of Major-General, and has been appointed Director-General of Medical Services.

VICTORIAN BRANCH NEWS.

Section of Industrial Medicine.

The next meeting of the Section of Industrial Medicine of the Victorian Branch of the British Medical Association will take place on Tuesday, September 20, 1955, at 8.15 p.m., at the Footscray and District Hospital. A symposium entitled "Surgical Problems Associated with Industrial Cases" will be presented by the honorary staff. All members of the Section are cordially invited to attend.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

UNEXPLAINED DEATHS: A PLEA FOR ACCURACY IN REGISTRATION.

[From "Diseases of the Heart", by Andrew Ross, M.D., New South Wales Medical Gazette, November, 1871.][1]

THERE are scores of people I have no doubt in the colony as fully conversant with the subtle nature of many common and energetic poisons, as there are abundance of others equally well versed in hocussing or the art of doctoring grog. I say then the more numerous such cases happen the more they tend to arouse (whether well or ill founded I don't attempt to say) a latent suspicion of something more common than disease of the heart—unless it can be substantially proved that climate, habit, occupation, or food of the people has anything to do in accelerating such cases, occurring too, not only during the extreme heat of summer, but even in the midst of the cool months of winter. The question may be asked, how are these deadly enemies to be obtained? Easily enough because they are distributed in N.S.W. without let or hindrance. Only look at strychnine—a poison almost as common in the bush among the shepherding class as tobacco. But it is becoming fashionable to refer all things seemingly to this popular archangel called "disease of the heart", so that I will now probably be considered out of order in thus herein alluding to the matter. Look the question sternly in the face and I ask is it not sufficient to arouse some attention—especially that of the medical jurist.

When organic disease is absent, or but faintly developed, then I think in all cases of sudden death, nothing short but the result of a chemical analysis should be received as conclusive evidence. These sudden deaths suggest grave reflections on the insecurity of human life in the bush, especially under the present system adopted in registering the cause of death. In a very large majority of these registrations no certificate is ever obtained, and as seldom ever demanded, consequently life is left almost quite unprotected and the

[1] From the original in the Mitchell Library, Sydney.

Registration Act in the interior becomes not only a perfect farce but a useless waste of public money. Take for instance the death of any child that may die suddenly from convulsions, apoplexy, perhaps teething or some other natural cause. Deaths from such causes may be afterwards registered without any trouble or even a certificate, and while all this may be regularly enough done, the sad disaster all the time might very probably have resulted from secret poisoning. Here is a sample of those loose meaningless verdicts culled from a late edition of a country newspaper: "That the immediate cause of death was the stoppage of the heart's action, consequent on nervous exhaustion, the result of habitual intemperance." What mortal on earth can understand such a rigmarole finding, or is it apparent whether it was the stoppage, the exhaustion, or the intemperance that produced death, for surely it did not take three when one was enough. In plain English, it was the latter or the blood poison—for don't the stoppage of the heart's action occur in every kind of death? If the grog was not the cause, then probably the action might not have been disturbed. A man falls a tree with an axe, but let the axe alone and the tree still remains. It was not the axe then but in reality the man that fell the tree. It was not the heart that ceased to beat, but the grog probably that prevented it, therefore the grog wholly and solely was responsible for the death.

'Tis a pity a spade is not called a spade and everything else by its proper name.

Correspondence.

CHEMOTHERAPY WITH ANTIBIOTICS AND ALLIED DRUGS.

SIR: I should be pleased if you would draw to the attention of your readers the following inaccuracies that appear in the National Health and Medical Research Council Special Report Series No. 6 on "Chemotherapy with Antibiotics and Allied Drugs", which was recently distributed throughout Australia.

Page 8, line 34:

Delete sulphamethazole (syn. Gantrisin, Urolucosil).

Insert sulphafurazole (syn. Gantrisin).

Page 10, lines 1, 8, 11, 15:

For sulphamethazole *read* sulphafurazole.

Page 33, lines 39-42:

Delete remarks on sulphamethazole.

Page 90, line 23:

For 110 units *read* 1, 10 units (see also Table 2).

Yours, etc.,

A. J. METCALFE,
Chairman, National Health and Medical Research Council.

Department of Health,
Canberra,
August 24, 1955.

OUT OF THE PAST.

SIR: "Medico" will have a lively rival in the Minister for Health if the account was for treatment in a public ward!

Yours, etc.,

"HONORARY MEDICO."

Lismore,
New South Wales,
August 20, 1955.

PRIMARY ACUTE INFLAMMATION AND PRIMARY IDIOPATHIC INFARCTION OF THE GREAT OMENTUM.

SIR: I was very interested to read Mr. Thomas Rose's article on acute inflammation of the omentum (August 20, 1955), and particularly his reference to recurrence being unknown. The following case may be of interest. Mrs. K., aged forty, presented on October 31, 1952, with severe abdominal pain, forty-eight hours' duration. At laparotomy an omental mass attached to anterior abdominal wall, in the left iliac fossa, was found; there was no other pathology, and the inflammatory mass in the lower portion of the omentum was excised. Convalescence was rapid and uneventful. Three weeks later she again complained of increasing abdominal pain for two days. On examination there was a tender mass to the left of and above the umbilicus. Chemotherapy was instituted, with improvement of symptoms in twenty-four hours. Conservative treatment was continued and the mass was no longer palpable on the fifth day.

Yours, etc.,

B. DUFFY.

137 George Street,
Bathurst,
New South Wales.
August 22, 1955.

Australasian Medical Publishing Company, Limited.

ANNUAL MEETING.

THE annual meeting of the Australasian Medical Publishing Company, Limited, was held at The Printing House, Seamer Street, Glebe, New South Wales, on August 20, 1955, Sir Henry Newland, the Chairman, in the chair.

Directors' Report.

The report of the Directors of the company was as follows:

The Directors submit their report for the twelve months ended June 30, 1955, together with the balance sheet as at June 30, 1955, and the profit and loss account for the twelve months ended June 30, 1955.

It is with regret that we report the death at Sydney on January 20, 1955, of Dr. Arthur Madgwick Davidson, who had been a director and a member of the company from August, 1934, to January, 1947, and who did much to assist in the progress of the company.

THE MEDICAL JOURNAL OF AUSTRALIA continues to increase in size and circulation, and contributions have covered a wide field. It is a matter for congratulation that during the year Dr. Mervyn Archdall completed twenty-five years of valued service as Editor of the journal.

A satisfactory result was obtained from the year's production of the printing and publishing department, and arrangements have been made for the payment of debenture interest for the year ended June 30, 1955.

Following the successful appeal of the company against the decision of the Council of the City of Sydney, as authority under the County of Cumberland Planning Scheme, not to permit additions to The Printing House, the Directors have under consideration plans for a western extension to the building. The extension will provide much-needed space for expansion and for amenities for the staff, and we hope to have the work completed in 1956.

The company's reserves are used in the business and we consider the state of the company's affairs is satisfactory.

Dr. W. L. Calov and Dr. A. E. Lee retire from office by rotation in accordance with the Articles of Association (Article 39). They are eligible and present themselves for reelection.

H. S. NEWLAND,
Chairman

July 12, 1955.

Election of Directors.

Dr. W. L. Calov and Dr. A. E. Lee were reelected to the Board of Directors.

COMPLIMENTARY DINNER TO THE EDITOR AND PRESENTATION OF PORTRAIT.

ON August 20, 1955, the Directors of the Australasian Medical Publishing Company, Limited, entertained the Editor and his wife at dinner at the Carlton Hotel, Sydney. Among those present were the members of the Australasian Medical Publishing Company, Limited, the members of the Federal Council of the British Medical Association in Australia, Dr.

Louis H. Bauer, Secretary-General of the World Medical Association, Dr. J. O. Mercer, Editor of *The New Zealand Medical Journal*, and Dr. Robert Scot Skirving, together with members of the medical profession who, through the years, had helped the Editor, either in editorial writing or in advice on special matters. The Chairman of the Australasian Medical Publishing Company, Limited, Sir Henry Simpson Newland, presided.

In proposing the health of the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, Dr. Mervyn Archdall, Sir Henry Simpson Newland used the following words:

> At a meeting of the Directors of the Australasian Medical Publishing Company held early this year, Dr. Archdall at the conclusion of his editorial report rather startled the Board by casually announcing that he had completed twenty-five years' service as Editor of the journal. The Board forthwith lavished their congratulations on him. Congratulations being the only cheap thing in Australia, they realized that their action could not rest there. He was invited to have his portrait painted, not, I hasten to add, at his own expense. A little later it was decided to entertain Dr. Archdall at dinner and to present the portrait on that occasion. The espousal of the Editor with the fortunes of THE MEDICAL JOURNAL OF AUSTRALIA and his cherishment of it for twenty-five years has led to this occasion taking the form of a silvern celebration of the nuptials of twenty-five years ago. This is in fact a wedding breakfast, and I trust that it will be celebrated with the jollity of such feasts. It is tendered to our Editor as a tribute for his service to THE MEDICAL JOURNAL OF AUSTRALIA, so valuable and so tireless. The Directors are happy that this banquet is graced by guests who are linked to our Editor by friendship, or by the good repute in which he is held. Dr. Archdall is a man with chivalrous instincts and fine feelings, admirable qualities in the Editor of a weekly medical journal, but which would unfit him for the editing of the agony page of a daily paper, with its toll of battle, murder and death, sudden or protracted. Those to whom our Editor is best known are aware that he is a man with a relish, a man with a true literary faculty as well as palate. In the words of John Morley, he has "a moral relish for veritable proofs of honesty". He lives his life with a relish. He relishes his work. He relished the arts and music and he sings a good song. He relishes a good joke, and more in charity than with relish, he has been known to laugh at a poor one of mine.

The toast was supported by Dr. Robert Scot Skirving and Dr. Allan Walker, and was drunk with enthusiasm. The Chairman then unveiled the portrait, which had been painted by Mr. Jerrold Nathan, of Sydney, and asked Dr. Archdall's acceptance of it.

In his reply, Dr. Archdall expressed his appreciation and gratitude to the Directors of the Company for their expression of confidence and goodwill. He hoped that those present liked the portrait as much as he did, and he hoped that it would find a permanent resting place in the Board Room which would be built as part of the western extension of The Printing House. After expressing his appreciation of the honour done him by the presence of Dr. Skirving, Dr. Archdall named in turn those who had helped him in the editorial work in many ways. He referred especially to three from Victoria who had been unable to be present—Emeritus Professor W. A. Osborne, Dr. Reg. S. Ellery and Dr. Byron Stanton. He said that a man who sat to have his portrait painted was in somewhat the same position as a patient facing an abdominal laparotomy. He did not know what the surgeon-artist was likely to find and whether he would display all that he had found. He was grateful to Mr. Nathan in that, if he had found any reprehensible or unworthy characteristics, he had not displayed them on the canvas.

BUFFET LUNCHEON AND LAYING OF THE FOUNDATION STONE OF WESTERN EXTENSIONS AT THE PRINTING HOUSE.

ON Tuesday, August 23, the Directors of the Australasian Medical Publishing Company entertained some 120 members of Congress at a buffet luncheon at The Printing House on the site of the new additions which are shortly to be made. Visitors were received by the Chairman of Directors, Sir Henry Simpson Newland, and other members of the Board, and also by the executive officers and members of the staff of The Printing House. After luncheon Sir Henry Newland

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED AUGUST 20, 1955.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	5(3)	7(6)	2(2)	1	..	..	..	..	15
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	1(1)	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	1(1)	..	..	..	..	..	..	..	1
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	1	14(10)	3(2)	..	..	2	..	..	20
Diphtheria	6(4)	3(2)	2	1(1)	1(1)	..	..	..	13
Dysentery (Bacillary)	..	6(5)	1(1)	..	3(3)	2(1)	..	..	12
Encephalitis	..	3	1	..	..	..	..	..	4
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	..	..	..	..
Infective Hepatitis	47(22)	85(39)	..	4(3)	5	..	1	..	142
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	1(1)	..	..	..	..	..	1
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	3	3(2)	1(1)	..	..	1	..	..	8
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	1(1)	..	..	5(1)	..	..	..	..	6
Puerperal Fever	..	..	..	..	..	1	..	..	1
Rubella	..	16(10)	..	2	6(6)	..	..	..	24
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	21(13)	18(9)	32(9)	3(3)	3(2)	..	..	..	77
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	1(1)	..	..	1(1)	..	..	..	2
Trachoma	..	..	..	..	3	..	..	..	3
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	22(13)	18(11)	8(5)	12(8)	6(5)	4(3)	1	..	71
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

laid the foundation stone of the western extensions and told those present that it was hoped that building would soon be started; the plans had been approved and a tender for building had been accepted. The wording of the foundation stone is as follows: "The Foundation Stone of the Western Extension to The Printing House was laid by Sir Henry Simpson Newland, C.B.E., D.S.O., M.S., F.R.C.S., Chairman of Directors of Australasian Medical Publishing Company Limited, on 23rd August, 1955." The company then inspected the several departments of The Printing House and had explained to them the processes carried out in each department. Among those present on the occasion were Dr. Louis H. Bauer, Dr. Talbot Rogers and Dr. J. O. Mercer. Dr. Talbot Rogers was attending the Australasian Medical Congress as representative of the Parent Body of the British Medical Association. There were also present Sir Charles Blackburn, President of the Ninth Session of the Australasian Medical Congress (British Medical Association) and Chancellor of the University of Sydney, with other representatives of the University, and sectional office-bearers of Congress, members of Branch Councils and a number of interstate visitors.

PRESENTATION TO EMPLOYEES WITH MORE THAN THIRTY YEARS' SERVICE.

ON Monday, August 22, 1955, an interesting ceremony was held at The Printing House. The Directors of the company with Sir Henry Newland, Chairman, met the employees for the purpose of recognizing the services of those who had been with the company for more than thirty years. Dr. W. L. Calov, Vice-Chairman, introduced Sir Henry Newland, who explained the object of the gathering and expressed the keen appreciation of the Directors at the excellent work which had been carried out over the years at The Printing House. Those with more than thirty years' service would be presented with suitably inscribed watches as tokens of appreciation and goodwill from the Directors. Each employee in turn was introduced to Sir Henry Newland and the presentations were made. The six employees who received watches were Miss Nellie Phillips, Mrs. Margaret Evans, Dr. Mervyn Archdall, Mr. J. A. P. Blacker, Mr. W. Souter, and Mr. R. Magrath. Mr. A. P. Dunlop, who was to have been among the recipients, was unfortunately absent owing to illness.

Obituary.

ARCHIBALD JOHN COLLINS.

DR. ARTHUR D'OMBRAIN writes:

"Beloved physician", that was Archie's name,
Of all his gracious qualities, the crown;
More than his leadership—his lasting fame:
A doctor, who would never let you down.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Cook, Warwick John, M.B., B.S., 1953 (Univ. Sydney), 102 Balfour Road, Rose Bay, New South Wales.

Eichel, Ferdinand, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act*, 1938-1955, 12 Penkivil Lane, Bondi, New South Wales.

Herz, Leopold Max, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act*, 1938-1955, Flat 8, 81 Roslyn Gardens, Elizabeth Bay, New South Wales.

Sproule, Brian Creighton, M.B., B.S., 1954 (Univ. Sydney), 19 Waverley Crescent, Bondi Junction, New South Wales.

Symes, Robert Matthew, M.B., B.S., 1954 (Univ. Sydney), Box 8, P.O., Tingha, 5N, New South Wales.

Deaths.

THE following deaths have been announced:

GUNDERSEN.—Sara Elisabeth Gundersen, on August 5, 1955, at Melbourne.

CARROLL.—William Carroll, on August 27, 1955, at Sydney.

PARRAMORE.—George William Parramore, on August 27, 1955, at Sydney.

Diary for the Month.

SEPT. 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 7.—Victorian Branch, B.M.A.: Clinical Meeting.
SEPT. 7.—Western Australian Branch, B.M.A.: Branch Council.
SEPT. 9.—Tasmanian Branch, B.M.A.: Branch Council.
SEPT. 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 19.—Victorian Branch, B.M.A.: Finance Subcommittee.
SEPT. 20.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 21.—Western Australian Branch, B.M.A.: General Meeting.
SEPT. 21.—Victorian Branch, B.M.A.: Clinical Meeting.
SEPT. 22.—New South Wales Branch, B.M.A.: Clinical Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all contract practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 *per annum* within Australia and the British Commonwealth of Nations, and £6 10s. *per annum* within America and foreign countries, payable in advance.